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The alimentary tract

Alfred Ernest Barclay



The Alimentary Tract

Roentgen rays (X-rays, etc.)
Geo. W. Holmes.
THE ALIMENTARY
TRACT

A Radiographic Study

BY

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*" There are more things in Heaven and earth, Horatio,
Than are dreamed of in your philosophy."*

" What a piece of work is man."

PREFACE TO THE FIRST EDITION

THIS little volume is, in essence, a thesis.* Sir Clifford Allbutt, Sir William Osler, Dr. R. Hutchison, and others who saw it were kind enough to suggest that it was worthy of publication, and these pages are re-printed with slight alterations and additions from the *Medical Chronicle* by the courtesy of the Editor, Dr. E. M. Brockbank. The chief alteration is the inclusion of Chapter VIII on the *Ætiology* of Gastric Ulcer, which was conceived and written after correcting the rest of the proofs of this volume. To Dr. J. Gow I am indebted for the Index, and to Prof. Dr. Gocht for the Bibliography.

March, 1913.

* Submitted for the degree of M.D., at Cambridge, in April, 1912, and placed prox. access. Horton Smith prize.

PREFACE TO THE SECOND EDITION

THAT there is need for a book on the radiosopic examination of the alimentary tract is evident from the call upon the first edition of this volume under the title of "The Stomach and Œsophagus," which was no more than a thesis, practically unaltered for publication. In the present volume this thesis still forms the groundwork, but it has been considerably enlarged and altered in accordance with extended experience.

When dealing with such a subject as this, that is still in its infancy, it is quite impossible to lay down the law; one can do no more than give the lines on which one does one's own work. The mechanism and nervous control of the alimentary canal are practically unexplored and full of problems waiting to be solved. Little by little new facts are being brought to light, and one hopes that in this pioneer work the radiologist will take his part. I hold no brief for the infallibility of the x -ray method, or for the radiologist as the one and only guide to the solution of the problems of disease within the abdomen. But the trained radiologist, armed with the bismuth method, possesses advantages for extended observation and investigation that are not usually held by clinicians. On the other hand, he works in a department and cannot avoid departmental bias—it is the man who can combine his clinical knowledge with his special facilities for extended observations, co-ordinating his results, looking for and learning from his failures, who will be the most successful in this work. This volume, therefore, is an attempt to bring my own departmental knowledge to the assistance of those who care to use it.

When the thesis was compiled some three years ago, one still clung to the original conception of the alimentary canal divided into its anatomical sub-divisions. One thought of a gastric ulcer, an hour-glass stomach, a spasmodic hour-glass stomach, a duodenal ulcer, etc., as definite clinical entities. The lesions of each anatomical division were considered as

being intrinsic or belonging to that compartment only. In writing up the thesis one was gradually driven to the conclusion that this view was wrong, and that the diseases in the stomach and duodenum are, more often than not, end results only. Therefore the chapter on the ætiology of gastric ulcer was inserted. During the last eighteen months more and more evidence has come to hand in support of this view, both in my own work and in the writings of surgeons and radiologists. One is now forced to consider the alimentary tract, from the cardiac orifice to the pelvic colon, as one organ, for the nervous system is so intimately connected that referred signs and symptoms are the rule rather than the exception.

Having found evidence of a duodenal ulcer or some other lesion, one is no longer content with this as a diagnosis. One would like to go further and detect the primary cause; for I am convinced that the large majority of the ulcerations met with in the stomach and duodenum are secondary manifestations, and are the direct result of disease in other parts. And, having arrived at these conclusions, and being more and more impressed by them every day, the shortcomings of such a work as this that I have written are all too patent. One knows that it is but the elements of an abstruse problem, a tangled skein, that will rapidly be unravelled in the next few years; the work of one departmental observer must necessarily fall far short: the solution will be the combined work of surgeons, physicians, radiologists, physiologists, and anatomists.

The work embodied in this volume is based on fluoroscopy, and, although radiograms are taken, the diagnosis is invariably made on the observations of the shadows seen on the screen. One is frequently asked to "take a photo of the stomach after an opaque meal," and there is still a widespread belief that a diagnosis can be made by taking a radiograph or a series of radiographs. Hence there is a tendency for radiologists to cater for this demand and aim rather at giving a pretty picture than a correct diagnosis. It is a great deal easier to be a radiographer, taking pictures of opaque meals, than a radiologist who interprets the shadows that he sees. And to do this correctly requires not only radiological experience, but

also the following up of cases to the operating theatre and post mortem room.

Friends have been most kind both in criticism and suggestion, and I owe much to them. To the staff of the Anatomical Department of the Manchester University, Professor Elliot Smith, Professor Wingate Todd, Mr. Stopford, and others I am indebted for much inspiration and help, especially in respect of the work on the normal stomach, while the staff of the Manchester Royal Infirmary have given me every possible encouragement at all times.

June, 1914.

War, wholesale slaughter, collapse of credit, and God knows what to follow in the wake, are upon us in Europe. Culture, art, and learning are overshadowed by a menace such as destroyed the ancient civilisations. Barbarity and brute passion are let loose, armed with the gifts of applied science. Science has forged the weapons that are being turned against her, and who knows where it will end? Will civilisation stand the shock? It is in despondency that I pen these last lines, but—

“There shall never be one lost good! What was, shall live as before;

“The evil is null, is nought, is silence implying sound.

In the shadow of this calamity I have thought it best to send out this work as it stands. Several chapters I had intended to re-write again and enlarge. The ideas embodied in the chapter on the large intestine have only recently taken shape and they are far from complete even in the mere writing up.

As the volume now covers the whole alimentary tract, it has been suggested to me that the title of “The Stomach and Œsophagus” should be altered to cover the ground included in the volume, and this I have accordingly done.

August, 1914.

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CHAPTER I.

INTRODUCTION.

THE diagnosis of gastric and intra-abdominal lesions is perhaps the most intricate and difficult branch of medicine, and even in experienced hands the condition found post-mortem or on the operating table is often very far different from that which was suspected from a full consideration of all the various signs, symptoms, and clinical tests available. Therefore the importance of any new method that will be of assistance in the diagnosis of these cases cannot be overestimated. The application of the *x*-rays in the diagnosis of these conditions has been productive of excellent results in this respect, and is undoubtedly a great advance not only in the conception of the normal stomach and its working but also in the detection of various pathological conditions.

In the early days of *x*-ray diagnosis one expected that by merely seeing the shape of the shadow of the food in the stomach a diagnosis could at once be formed. It was only after a number of failures that one was driven to the obvious conclusion that a thorough study of the normal stomach was essential. A better knowledge of the limitations of the method must be arrived at before undertaking the examination of the pathological cases.

The difficulties of interpreting the shadows cast by the bismuth meal in the stomach are great; for not only is the organ absolutely different in shape during life from the pictures one would expect from the study of anatomy, but also even slight alterations in tonic action will produce a picture that *appears* to be very different. Again, the conditions found at operation were often quite the reverse of what one expected from the *x*-ray examination. For instance one reported, after *x*-ray examination, a stomach of normal size, and the surgeon at the operation found a large flaccid sac and *vice versa*.* And in the study of the normal stomach great

* For explanation, see pp. 40 *et seq.*

difficulty was experienced, for it was found that even in healthy subjects the organ apparently varied to an amazing extent, not only in shape and position but also in the manner in which the muscular walls appeared to act. Another difficulty was presented by the fact that the pictures seen when the patient was in the upright position differed widely from those obtained when the patient was examined lying on the couch. Moreover, one did not necessarily see the same picture of the stomach each time one observed the patient, even under precisely similar conditions, and one was also occasionally confronted with such phenomena as that of a healthy subject whose stomach seemed to conform to all one's ideas of what a healthy organ should be on one examination, while on the following day a picture that seemed to bear little or no resemblance to the previous one was seen.

During the first two or three years at this work it was often a matter of speculation as to whether one would be able to obtain *x*-rays of sufficient power to penetrate the abdomen of any but the thinnest patients, and in a great number of the early cases one had the utmost difficulty in obtaining even a rapid glance of the shadow before the force of the currents used wrecked the *x*-ray tube. It was very seldom that one could study the pars pylorica. Moreover, it was only when the apparatus was working at its most efficient pitch, and when the *x*-ray tube happened to be in exactly the right condition, that a radiograph could be taken. Even then the exposure had to be of such lengthy duration that all detail was lost owing to the peristaltic movements of the walls.

Latterly, however, one has been able to rely upon obtaining a satisfactory screen examination in nearly all cases, and it is only in the study of the pars pylorica that one could often have wished for greater clearness in the screen image. Radiographs have often been taken, but they have seldom, if ever, revealed greater detail than could be made out by the screen examination, owing to movements during the lengthy exposures that were necessary. The introduction of the intensifying screen, however, has reduced the exposures to a matter of a second or so, with the result that whenever it was not possible to see exactly what was going on, a picture could

be taken that would be of considerable assistance in the diagnosis.

Such are some of the difficulties which have beset the earlier stages of this work, but, apart from the technical difficulties, one conclusion stands out with great clearness; no one picture can be taken to represent the normal stomach, and no detailed study of a small number of normal subjects can be of great value in itself. It is only by the consideration of a very large number of cases that a clear conception of the possible variations of the normal stomach can be attained, and it is only after this knowledge has been obtained that the value of the *x*-ray method of examination in the diagnosis of pathological conditions of the stomach becomes of the highest value.

The whole alimentary canal, but especially the stomach, is an exceedingly sensitive muscular organ, and spasmodic conditions have been found (and during the last few years recognised) in a large proportion of pathological cases. These spasmodic contractions have been a source of much trouble in diagnosis, and it was not until I realised that they entered into and complicated almost every active lesion involving the mucous membrane that I appreciated their importance, not only in complicating the diagnosis but in interfering with the gastric functions, in many cases causing complete functional biloculations of which no suggestion was found at the operation. It was found that the extent and severity of the spasmodic element of organic lesions did not bear any definite relation to the size or appearance of the ulcerations, and the suggestion is that the severity of the spasm depends rather on the irritability of the ulcer than upon any other factor, for sometimes a large ulcerated surface (especially on the lesser curvature) gave rise to practically no spasmodic contraction, while even small ulcers (especially of the greater curvature) sometimes gave rise to such powerful and persistent spasmodic contractions that one was confident that the surgeon would find a typical cicatricial hour-glass contraction.

In this small volume, therefore, I propose to deal chiefly with the explanation of the various difficulties met with in the

normal and pathological cases examined, and to lay down the signs on which the diagnosis of morbid conditions may be given. In every case full use has been made of the clinical history and such other evidence as was available before giving an opinion, and although one is so thoroughly convinced of the enormous value of this method—especially in indicating those cases that are suitable for operation—one must urge that the *x*-ray method is only one of the means at our disposal, and that if we do not use all the evidence available, the use of this method by itself will lead to mistakes in diagnosis, and we will be doing less than justice not only to the means of investigation at our disposal but also to the patient.

I am quite conscious of having received many suggestions from writers on the subject but, as will be seen, the work is based almost exclusively on my own examinations. I have therefore omitted a historical outline and summary of the work done by others in this branch of medicine.

It was Cannon who first initiated the study of the intestinal movements in dogs and cats by means of giving large doses of bismuth, but as applied to the examination of the human alimentary canal, Rieder of Munich was the pioneer, for it was he who first demonstrated that the large doses necessary for these examinations were quite harmless, although previously Roux and Balthazard had attempted diagnosis by means of 5 drachm doses. Many observers have written on the subject but the following names stand out most prominently:—Holzknecht and Jonas of Vienna, Jolasse, Leven and Barret, Haudek, Groedel, Rosenthal, Kaestle, Hertz and Case.

My own work on this subject commenced in 1906, and since then I have availed myself of every opportunity of examining both normal and abnormal subjects. I have notes of nearly 900 patients examined* and there are many others of whom I have no records, either owing to the rush of

* Up to December, 1913, I had notes on about 2,200 cases. A proportion of these were examined in conjunction with my partner, Dr. Bythell.

hospital work, or, as in many of the normal cases, because nothing worthy of note was found at the examination. As a routine practice each patient is observed as he takes the bismuth food and at frequent intervals until I am satisfied as to the rate and manner of emptying. According to the impression received from this first examination instructions are given for feeding the patient so many hours before examination on the following day so that every observation of abnormality can be confirmed. Indeed I think it is of the utmost importance to confirm every observation no matter how clear the signs may appear, so that the actual number of observations must be very large.

CHAPTER II.

TECHNIQUE.

1. FUNDAMENTAL PRINCIPLES.

The *x*-rays penetrate all substances to a greater or lesser extent, the resistance that is offered to their passage being approximately in direct proportion to the specific gravity. The walls of the alimentary canal do not differ from the rest of the abdominal contents in this respect, and consequently they give no distinctive shadow on the fluorescent screen or radiogram.

The method therefore depends on the filling of the alimentary canal with some substance that differs as widely as possible in density from that of the tissue structures, *i.e.*, something very heavy, such as a bismuth or barium salt, or by inflating with air or gas. In the former case the contents obstruct the rays and therefore a dark shadow is thrown upon the fluorescent screen, whereas in the latter, the air allows the rays to pass more freely and therefore the fluorescent screen is more brilliantly illuminated. This latter method is, however, of very limited value.

2. METHODS OF EXAMINATION.

We have the choice of radiography and radioscopy as our method of examination, and in my opinion there is no comparison. Radioscopy is the method of necessity. The examination should be undertaken with the patient standing with his abdomen against the fluorescent screen, and the passage of the first mouthfuls should always be carefully observed from the lower third of the œsophagus till the lower part of the stomach is outlined. I have found that examinations of the stomach made in the upright position yield infinitely more information than those conducted with the patient lying down, and as these latter examinations give a different picture which may be very misleading, I have

purposely omitted any description of them in order to avoid confusion.

Radiographs of the stomach are of comparatively little use except for demonstration purposes; they represent the picture at one particular moment only,* and give little indication as to how the stomach receives the food, etc. Radiographs are therefore expensive, and in many cases unnecessary luxuries except for demonstration purposes, but a good radiogram of the pars pylorica will often reveal more detail than can be made out on the screen.

Cinematographic radiography is, of course, a substitute for radioscopy, but, apart from the absolutely prohibitive expense, it can, at best, yield only a picture of one or two cycles of the stomach's movements. These may, and often do, vary from time to time. But even more important is the fact that palpation, radioscopy, cannot be practised. The same objections hold as regards "serial radiography," the taking of a rapid series of 40 or 50, 10×8 plates of the lower two-thirds of the stomach, and making deductions from superimposing and comparing the pictures obtained in this manner. To me it seems that the expense and inefficiency of these methods (because of the absence of palpation) are quite unjustifiable seeing that, at the present time, we know the danger of *x*-ray burns and are quite capable of guarding ourselves. Those who have suffered from *x*-ray dermatitis should be very chary of screen examination, but I hold there is no excuse for those who have not suffered this misfortune, in subjecting patients to the inefficiency of radiographic methods and to the expense of an examination that could be better, even if less theatrically, achieved by a competent observer with his own eyes. My own experience during the

* When it is wished to obtain a radiograph of one particular phase of this gastric movement, this can be done by watching several waves and timing them with a stop watch. It will be found that there is a constant time factor for each cycle, usually about 20 seconds. The same picture will therefore recur at intervals of 20 seconds and, having placed the plate in position, the exposure is made when the appearance we wish to record is due. We owe this suggestion to Dr. Thurstan Holland.

last seven years justifies me in stating that there is not the slightest need for the excessive precautions that some observers affect to think necessary. The pendulum swings from excessive carelessness, from which there are still many sufferers among the observers, to an excessive carefulness that is a menace to the utility of this extremely valuable (and reasonably economical) method of diagnosis.

3. PROTECTION OF THE PATIENT.

It is the soft rays, the ones which are easily stopped and are therefore useless for this type of examination, that produce *x*-ray dermatitis. For this reason the patients have worn a dressing-gown or other garment, and in practice this has been found quite sufficient protection, as not a single case of reddening of the skin has been noted, although many of the examinations—especially for demonstration purposes—have been very prolonged. Moreover, repeated examinations have been necessary in practically every case, some of the early ones being examined as many as eleven times.

4. PROTECTION OF THE OBSERVER.

From the outset—in 1906—it has been evident that a radiograph of the stomach contents, no matter how perfect, could be of little value in diagnosis, just as a photograph would be useless in depicting an ataxic gait. Therefore examinations have invariably been conducted by means of the fluorescent screen, and this has necessarily involved a great deal of exposure to the rays on the part of the observer. Great difficulty has been experienced in devising apparatus for self-protection that would yet allow of easy manipulation of the patient. When examining with the subject lying on a couch there was little difficulty, as one was not in the direct path of the rays, but for examination in the upright position the observer is necessarily in front of the tube. An additional and very heavy protective diaphragm was therefore made by the hospital staff of carpenters, and the quantity of rays that now penetrates is infinitesimal.

At all times *x*-ray proof gloves and apron have been worn and no dermatitis has been sustained. In the early days,

before the fluorescent screen was covered with efficient x -ray proof glass, irritation of the eyes was of frequent occurrence, but no permanent ill-effects have resulted.

During periods of heavy work, before the operator was properly protected, it was found that excessive weariness and mental inertia were the immediate result of a series of screen examinations, but, since the addition of the protective diaphragm already referred to, this effect has not been noted to the same extent. The only ill-results now encountered, even on the busiest days, being slight weariness and headache, which are probably not due to x -rays but to working in a dark ill-ventilated room in which the air is in a partially ionised condition. A ventilating fan was therefore placed in the wall, and now there is apparently no undue weariness even after the busiest morning's work.

5. POSITION OF THE PATIENT.

(a) *In œsophageal cases.* The upright position is the easiest and most useful as well as the most natural for œsophageal cases. In the direct antero-posterior position the vertebral column and heart form a very heavy shadow through which it is impossible to see the œsophagus clearly, but if the patient is slowly rotated so that the front of the right shoulder is against the screen, a certain angle is found at which these two shadows become separate; a comparatively clear space representing the posterior mediastinum is opened up, and it is through this space that the œsophagus passes. In this position the whole œsophagus is seen, from the pharynx to the cardiac orifice.

(b) *In gastric cases.* It was at once apparent that posture had an extraordinary effect on the shadow of the gastric contents, and it became a matter of choice whether to examine the patient standing or lying down; the limitations of time preventing a routine use of both positions. In many cases, however, both were employed, but the horizontal position practically never yielded any information that one had not already obtained, and its use was abandoned except for post-operative cases where the patient was too weak to stand and it was necessary to determine by what route the food left the

stomach. For this purpose the horizontal position sufficed, but it is not capable of yielding reliable data as to the stomach walls, and for this reason I think it futile to attempt ordinary x -ray diagnosis unless the patient is sufficiently strong to be examined in the upright position. Another point that determined the use of this posture was the fact that it is the usual position during digestion, and it is while this process is going on that the x -ray examination is made.

For watching shadows that are apparently due to food in the duodenum it is often of advantage to examine the patient lying down. The view of the duodenum is clearer in this position and also palpation is easier.

The best view of the stomach is obtained with the abdomen against the screen, but in certain cases it was found that the pyloric portion seemed to turn somewhat more backwards than usual and its shadow was thus foreshortened, but, by rotating the patient slightly, this portion came into full view. The pylorus itself is the most difficult portion to see clearly, not only on account of the relatively small quantity of food that it contains but also because of the superimposed shadow of the vertebral column.

(c) *In Intestinal cases.* For the examination of the intestine, especially the lower end of the ileum and large bowel, the recumbent position offers greater facilities for careful palpation associated with screen examination (radioscopic palpation), which is of such marked assistance in determining the existence of adhesions, etc., but it is always advisable to examine such patients also in the upright position, noting the effect of gravity on the shadows and comparing impressions obtained by palpation in both positions.

6. PREPARATION OF THE PATIENT.

Patients should be prepared for all abdominal examinations as if for an anæsthetic by means of purgatives, in order that the stomach may be empty and that there may be no shadows in the intestines to confuse the picture. This also makes the abdomen much more translucent to the rays, consequently the picture of the stomach full of bismuth has a much sharper outline than if no preparation had been made.

It is, however, advisable to have the preparation made the day but one before the examination, in order that all tendency to diarrhoea may have passed off before the examination is undertaken. The effect of a purgative is to hasten the passage of the food, not only in the large intestine but also in the small bowel. These patients are frequently taking some bismuth mixture; this should be discontinued for a week at least before the examination.

No special preparation is of course necessary for œsophageal examination.

That the patient should be examined during an attack is one of the most important points. If he is quite well when examined, it is probable that nothing abnormal will be found; whereas when the symptoms are present we may discover definite indications of the cause of the trouble. For, in many cases, these are dependent on spasmodic contractions which are coincident with, and probably the actual cause, of the pain from which the patient suffers.

No food of any kind should be given for at least three hours before the examination, in order that, if there is no obstruction, the stomach may be examined when it is quite empty.

7. FOODS USED.

(a) *In œsophageal cases.* The same food stuffs have usually been employed in œsophageal as in gastric cases, but the consistency has been varied according to the patient's statement as to the character of food with which he has difficulty. Cachets and capsules of bismuth have been discarded, as it was found that they gave quite unreliable evidence; in a healthy subject a gelatine capsule was delayed half an hour at the cardiac orifice in spite of copious draughts of water, while a delay of some minutes was sometimes seen behind the aortic arch. The passage of a solid bougie was watched in one case, but its use appeared to be so dangerous that I have never again employed this method.

(b) *In gastric examinations.* The essential feature of the food is that it should be of sufficient difference in specific gravity from the abdominal contents to cast a definite shadow.

This is attained by embodying in it a salt of one of the heavy metals. Bismuth subnitrate was used in the early cases, as much as 4 ozs. being taken with no ill-effects. Reports of cases of poisoning were published* and the carbonate of bismuth was subsequently used. The oxychloride of bismuth (introduced by Dr. A. F. Hertz) was tried, but I could find no appreciable difference in the behaviour of the stomach. Theoretically the carbonate of bismuth should be neutralised by the hydrochloric acid of the stomach, but in practice this is not found to be the case.† In fact in all the earlier observations of hypersecretion this salt was used and no evolution of CO_2 was noted, while on giving a dose of sodium bicarbonate it was at once apparent, from the increase in the size of the magenblase, that quantities of CO_2 were being given off. The oxychloride had, moreover, a tendency to settle out and to give a gritty taste to the food.

Since 1911 specially prepared barium sulphate has been employed in the greater number of cases; with this salt there are no theoretical disadvantages and the cost is approximately one-eighth of that of bismuth salts.

My own impression of these various salts is that the bismuth oxychloride and barium sulphate are absolutely alike and that both of them have a slightly stimulating action on the stomach, whereas the bismuth carbonate is either inert or is a little depressing to the activity of the gastric muscle. The difference is negligible from the practical point of view except in one type of case, *i.e.*, duodenal irritation. My practice

* At least two cases of poisoning occurred in America, but I have been unable to trace the references. Bennecke and Hoffman (*Münchener Medizinische Wochenschrift*," 1906, No. 19) recorded a fatal case, the symptoms being suggestive of nitrite poisoning, and nitrites were found both in the blood and pericardial fluid. Later Bohmie proved that the administration of bismuth sub-nitrate was followed by the appearance of nitrites in the fæces and urine, but not in the blood.

† In a series of experiments with test meals that had been recovered it was found that the addition of bismuth carbonate did not produce any evolution of CO_2 . On testing the acidity, however, it was found that there was a distinct drop although the acid was by no means neutralized by the carbonate of bismuth.

now is to use the barium sulphate in all cases, but when the picture of duodenal irritation is seen, in my confirmatory observations I use bismuth carbonate food for my opaque meal. There is little difference in the shadow cast by barium and bismuth although it sometimes seems that that of the bismuth food is rather sharper than the barium.

The actual quantities employed are not of vital importance and my nurse makes the meal by rule of thumb. It works out at about $2\frac{1}{2}$ ounces of bismuth carbonate, or 3 ounces of the barium sulphate, and bread and milk to the half pint. For stout patients rather more of the salt is put into the mixture. It is essential that the bread should be free from crusts and that there should be no large lumps. The bread, freed of crusts, is cut into fairly small pieces and the hot milk poured over it. The barium, or bismuth, is then well stirred in as it is added, and in the process all lumps of bread are broken up. Sugar is added in the form of a syrup and this makes a considerable difference to the palatibility—which is a point of great importance.

Porridge has been employed in some cases and offers neither advantages nor disadvantages.

Other excipients, such as mucilage of tragacanth, pounded biscuits, cake, bread and butter, mince meat, vegetables, etc., have been tried with more or less success, but the composition of the food seems to be of no practical moment, provided it does not nauseate the patient. The addition of raspberry syrup has been of some service in obviating this difficulty, but it is seldom that patients find the crude mixture too distasteful. Bread and milk, flavoured with plenty of sugar, is, on the whole, the best and has been employed as routine.

No fixed quantity has been given, but sufficient has been used in each case to dilate the potential cavity of the stomach to such an extent that the condition of the walls could be deduced. The quantity, therefore, has varied from 2 ozs. to $\frac{3}{4}$ pint—in one case of delusions as to the shape of the stomach a patient took 4 pints of custard mixed with bismuth carbonate (8 ozs.) with no ill-effects.

It is quite unnecessary to give purgatives in order to clear out the bismuth or barium after the examination. In large

doses these salts pass through unaltered and give rise to neither constipation nor diarrhoea.

The double bismuth meal, as suggested by Haudek, is of value in saving time. The patient takes a bismuth meal six hours before and is then again fed with bismuth at the examination. This method is very useful for at once obtaining evidence of the rate at which food leaves the stomach, but it has the very obvious disadvantage of giving an abdomen that contains a multiplicity of shadows which, in many cases, it is impossible to disentangle with certainty. At the confirmatory examination, 24 hours later, I employ this method, modifying my instructions according to the indications of each case as to the time at which the food is to be given. When it is the rate of emptying of the stomach that is in question the food is ordered to be taken some hours before, while in others one wishes to find the bulk of the second meal in the small intestine or near the ileo-cæcal valve, in which case the food is ordered according to the indications. The details of a routine examination are dealt with in Chapter III, p. 19.

(c) *In intestinal cases* the same opaque foods are employed as in gastric examinations. In the large intestine, however, except for observations on constipation, it is more usual, and far more reliable, to employ opaque enemata. These are made up with 6 or 8 ounces of barium sulphate suspended either in mucilage or other medium.* The addition of a little alcohol is said to help the suspension. My own preference is for boiled cornflour into which the barium is carefully stirred. This forms quite an effective suspension and contains no lumps if it is properly prepared. The enema is allowed to run in by gravity from a funnel through a good-sized rubber tube. The ordinary rectal tube may be used but it tends to block and we now employ a specially made cannula for the purpose. This is a straight metal tube of

* A prescription given to me by Dr. A. F. Hertz is :—

℞ Barium Sulphate	℥ ii
Mucilag Acaciæ	℥ ii
Spir. Vini Methylat.	℥ i ss.
Aq. ad	. i cc.

half an inch diameter fitted with a round-ended rod which is withdrawn after the cannula has been inserted. An olivary enlargement on the end of the cannula prevents it from being extruded.

8. THE IMPORTANCE OF EXAMINING THE WHOLE TRACT in every case cannot be too strongly insisted on. Referred pain and referred symptoms in the abdomen are the rule rather than the exception. It has been stated, by Moynihan I believe, that the most frequent seat of a gastric ulcer is the right iliac fossa; an epigram that contains more than a grain of truth. Apart from these referred symptoms, there are cases where patients give very inaccurate descriptions of their symptoms, and it is only after we know something of what is actually taking place that we can get a true picture of the pain and discomfort from which the patient suffers. This week, for instance, I have seen a case labelled clinically pyloric obstruction in which there was very marked oesophageal obstruction, while in another patient the symptoms pointed to obstruction of the splenic flexure, and the actual lesion was a growth that involved also the stomach beyond any hope of operative interference. (Fig. 49.)

9. SPECIAL METHODS.

(a) *Physical methods.* *Radioscopic palpation*, if one may so call the co-ordination of the senses of touch and sight, is extremely valuable. Practice in clinical palpation educates the tactile sense and it becomes very highly developed. In radioscopic palpation, education of the co-ordinated senses is even more essential, especially as, in examining the stomach, the patient is in the upright posture—a position in which we are not in the habit of palpating patients. The locating of a point of pain on deep pressure at one definite point where a spasmodic contraction, or increase of contraction, is noted as the result of pressure: the sense of resistance that one can see is due to the abdominal wall; the resistance that comes only when we get so deep that we are moving the shadows in the duodenum; the fixity of some portion of the stomach or intestine; the indefinite thickening felt over the

cicatrix of an hour-glass stomach; the clinically indefinite tumour that takes shape and meaning when felt in relation to easily moved bismuth shadows that are in relationship to it; these are but suggestions of some of the points that arise and from which the most valuable deductions may at times be made.

As applied to the small and large intestine this radioscopic palpation is even more valuable and requires less education, for the patient is lying down and the abdomen is more freely relaxed.

For this form of examination the necessary protection is a great inconvenience, yet on no account should the gloves be laid aside. It is, however, a great advantage to have the pads of the finger tips cut away. A wooden spoon (as suggested by Holzknicht) is an assistance—but it cannot feel. Where it is merely manipulation of the shadows that is needed, this very useful accessory will do the work, and it has the advantage that a plate can be taken of the field exposed by the spoon and yet the implement throws no shadow on the plate. This is perhaps of greatest use in locating and examining the appendix.

In manipulating the bismuth shadows it is very easy to mistake displacement of the contents of an organ for displacement of the organ itself and this source of error must be guarded against.

Abdominal massage, either during the examination or during a short interval, is most useful. I started the practice in 1909 and it has been of the greatest service. Spasmodic contractions will usually yield to massage, to some extent at any rate, and this fact is of considerable assistance in determining whether alterations in outline are the result of organic lesions or of purely spasmodic contractions, or whether, as frequently happens, there is a combination of the two factors.

Inflating the stomach by means of sodium bicarbonate and tartaric acid, distends the fundus, and has occasionally yielded information as to this portion of the cavity, but, owing to the impossibility of excluding gastric ulcer, it has seldom been used.

In the early cases, when good definition of the greater curvature could not be obtained, I found that *injecting air per rectum* was of some value, and in a few cases this method showed up small irregularities that would otherwise have escaped notice.

A mixture of bismuth and lycopodium has been tried for more clearly demonstrating the level of fluids, but its use was unnecessary and it has been abandoned.

Capsules made of gold-beater skin containing bismuth have been given, in order to test the digestive power of the gastric juice (suggested by Schwarz). I found that it was not possible to determine at what time the capsules ruptured and also that very frequently they were broken during deglutition. As no other bismuth could be given until the observations on these capsules were completed their use has been abandoned. This difficulty is overcome by Haudek who suggested that they should be given with the first portion of the double bismuth meal.

(b) *Actions of drugs.* The only drugs that have been examined to any extent have been sodium bicarbonate, belladonna, valerian, asafoetida, and alcohol.

Valerian and asafoetida have been found to diminish tonic action in proportion as patients found them distasteful. Those who disliked the smell or taste most showed the greatest loss of tone. Alcohol, on the other hand, tended to increase tonic action, the increase being more or less proportionate to the patient's appreciation of the alcohol (whisky) used in the experiments.

Belladonna acted in an uncertain manner. In some cases of spasm it caused complete relaxation, while in others it had little or no effect. In one or two cases of excessive peristalsis this symptom was checked by its action. No cases of excessive secretion have been tested with this drug as yet.

Sodium bicarbonate has also been found to check excessive and painful peristalsis in a wonderful way in a proportion of cases, while in others it has produced no effect. I have also introduced the use of this drug for testing, in a very rough but handy manner, the quantity of free acid in the stomach. In healthy subjects there is well-marked evolution

of carbon dioxide, but in every case of marked hypersecretion there has been much more profuse production of gas.

10. APPARATUS.

The simpler the apparatus the better—all one's attention is needed for observation and the elaborate and intricate appliance that gives every conceivable mechanical movement is, I believe, a mistake. My own apparatus consists of a tube stand in which the up and down movements and the control of the diaphragm are operated from an arm that extends forwards and is within easy reach. The screen is counterbalanced on a *separate* stand, on the platform of which the patient is placed. This screening stand moves very freely on castors and during the examination it is moved as required by the observer's feet. The observer sits on a bicycle saddle seat with his feet on the screening stand; and the patient is so far above him that it is easy to palpate the abdomen. There are several dark red lights in the room and these are connected to the *x*-ray switch board which is mounted on a trolley and stands close to the observer's right hand. He therefore has the whole control in his hands and has no need of any assistance except from the nurse, whose one duty is to feed the patient. A simple modification of the lighting is, that one of the lacquered ruby lamps has a clear patch that allows a stream of light to strike on the desk where the notes lie. This enables one to read the notes and add to them without turning on the ordinary lights of the room. I prefer red lights to the blue so commonly used, because red is the complementary colour to the green of the fluorescent screen.

CHAPTER III.

ROUTINE EXAMINATION.

THIS chapter is written with the intention of showing how a routine examination can be made with comparatively few observations, and yet devised so as to miss nothing of importance. It has been evolved from the experience of the limited and cut-up time that is at one's disposal when work is divided between institutional and private practice. One readily admits that it is not ideal, but it gives good results. I do not wish to suggest that my own routine variations of the bismuth method of examination are the only way of arriving at a diagnosis that is correct. Although a little cumbrous, it has the advantage of giving confirmation of first impressions. This is most important, for, as in other methods of diagnosis, it is most unwise to jump at a diagnosis, and I always avoid giving an opinion until I have had the opportunity of confirming my observations, even in the most obvious cases.

Haudek's double bismuth meal, *i.e.*, the patient fed a definite number of hours before the first examination is started, I have tried but do not employ it for routine work, as there have been few cases in which it has really saved time. It is useful in pyloric obstruction cases; but one never knows what type of case one will have to deal with, and one is badly handicapped if the examination is started with multiple shadows scattered through the abdomen which one cannot locate. It often means waiting till all this bismuth has been cleared out and then starting from the beginning again.

The preparation of the patient is discussed in Chapter II (p. 10).

A short history of the symptoms is taken, and special note is made of the time relation of food-taking to pain in order that one may examine, if possible, when the patient complains of symptoms. It is also important to note whether one is examining during or between attacks. In many

instances this is a great help in gauging the importance of appearances, especially if observations can be made both when the symptoms are severe and also when they are absent.

In the absence of notes of a physical examination the abdomen should be palpated, and in any case it is well to familiarise oneself with the feeling of an abdominal tumour before commencing the examination.

In œsophageal cases.

The patient takes a mouthful of opaque food of such consistency as he indicates will be difficult to swallow. He holds this in his mouth until one has made an examination of the aorta and posterior mediastinum. He is instructed to swallow it when one has secured the most favourable position for watching its progress. If no delay is noted with food that should be obstructed, according to the patient's statement, a drink of water or milk is given to wash out all traces of bismuth. Some dried or toasted bread-crumbs or a crust of bread are then given, and this is followed by another bismuth test. When there is no obvious cause for an obstruction one re-examines on the following day after a few doses of tinct. belladonnæ.

In gastric cases.

(1) *Normal.* After a glance at the chest and diaphragm movements, the first few mouthfuls of food are watched entering the stomach, and are followed carefully until the shadow has reached the lowest part. Perhaps a couple of mouthfuls may not be sufficient to canalise the empty stomach for a few minutes, and, to avoid waste of time, one gives a few more mouthfuls and watches the result. The lowest part is recognised by the rounded appearance and evident turning of the shadow to the right.

The patient then proceeds with his meal until he has taken as much as he can eat without discomfort. The state of the tonic action, the effect of the movements of respiration on the position of the lower border of the stomach, the peristalsis, and all other details are noted. Palpation is then employed and the tender part located. The stomach is moved and any

suggestion of adhesions looked for. Specially careful palpation is used over the pylorus, and an attempt is made, when a wave of peristalsis is about an inch from this point, to push some of the food through into the duodenum. Provided nothing abnormal has so far been detected, the patient now sits down for a time, and is brought up for observation at various intervals during the next hour or more until one is satisfied as to the manner of emptying. If one sees nothing but an indefinite, cloudy appearance in the lower abdomen at the end of an hour, and has noted no definite mass passing through the duodenum, the deduction is that the stomach is emptying in a normal manner and that there is nothing abnormal in the small intestine. The examination is now finished until the following day, and a packet of barium is given to the patient to be taken in bread and milk six hours before he is re-examined. Apart from taking this second opaque meal, he resumes his ordinary life, and eats whatever he is accustomed to. Of course no purgative is given, but, if the bowels are opened naturally, he should be instructed to note whether or not they contain quantities of barium.

At the *twenty-four hours examination* one should find the greater part of the shadow in the colon. If the bowels have acted, the descending colon, etc., will probably be empty. The whole of the six hours' food should have left the stomach, and the greater part of it should have passed into the cæcum. A few more mouthfuls of opaque food are given to see that the stomach gives the same appearance as before, and one palpates to see that the relative movements of stomach and colon are not in any way limited. If there is an unduly large collection of shadow behind the ileo-cæcal valve the patient is examined lying on the couch. In this position one can, as suggested by J. T. Case, draw up the cæcum and expose the terminal portion of the ileum and also the appendix. If this happens to contain any of the opaque meal it can be seen. The wooden spoon, suggested by Holzknecht, is most useful for this work, and one can take a plate with it in position, holding the cæcum up. Any point of tenderness on deep pressure is sought for as one manipulates the various portions of gut.

Cases of constipation are followed from day to day, but when there is a question as to the neoplasm of the colon an injection is much more reliable than deductions from the rate of progress of the food given by the mouth. This latter will give indications as to the part of the large intestine in which any delay occurs, but is quite unreliable in detecting organic obstructions, and in all such cases an injection should be given.

(2) *Duodenal and small intestine type of case.*

Proceed as in (1). If food is seen passing rapidly through the duodenum and accumulating in the small intestine, keep the patient under observation as long as possible and palpate the distended coils of small intestine, to see if the shadow is accumulated in one coil specially, or is spread more or less evenly through the coils. If the coils can be readily separated out and the shadows are seen passing into the region of the cæcum, one can be fairly sure that this appearance is due to the too rapid emptying of the stomach embarrassing the small intestine. To make certain of this the 24 hours' examination is made some two to three hours after a second opaque feed has been given. This feed is made up with bismuth carbonate instead of barium; the rapid emptying of a bismuth meal is much more suggestive than the same appearance after a barium feed, i.e., the bismuth is perhaps sedative, while the barium may be slightly irritating. Sometimes, in spite of the rapidity with which the stomach begins to empty, one finds in these cases that there is still quite a half of the food still in the stomach at this time: as if distension of the last coils of the ileum produced a reflex closure of the pylorus to prevent further overloading, i.e., a counterpart reflex to the gastro-iliac reflex (p. 98). Careful examination is made of any separate shadow in the duodenum and of the caput duodeni, and then some more food is given and observations are confirmed. As a rule this extra food seems to stimulate the whole tract, and again the stomach begins to empty fairly rapidly, but not so rapidly as before, although it is often possible to push large quantities into the duodenum.

More often, however, no food is found in the stomach, and,

having noted or radiographed the shadows in the right iliac fossa, one gives some more food and confirms one's observations.

In nearly all these cases there are accumulations in the ileo-cæcal region that call for investigation, and this is best done with the patient lying down as already described.

When there is a definite suspicion of delay in a certain coil of small intestine it is advisable to start *de novo*, if one does not obtain definite evidence by giving the meal a few hours before. For this purpose the patient takes mild purgatives for a few days, and then, after a preliminary glance to see that the abdomen is clear, one starts the examination again. Of course, it is very much to the advantage of the examination if the patient's symptoms are more severe when this examination is made, and in private practice my partner, Dr. Bythell, and I frequently instruct patients to come up for re-examination when the symptoms come on again, giving them a packet of barium to take a couple of hours before they are due to be seen. With hospital patients, such indiscretions of diet as the patient suggests are allowed until the attack comes on.

(3) *Pyloric obstruction type.*

When one detects the presence of retained food already in the stomach (p. 60) when the examination is started, it is not likely that, in one hour or so, one will see any food in the small intestine. If only a comparatively small quantity of retained food is noted, and the stomach is otherwise normal in appearance, except perhaps occasional powerful waves of peristalsis, one instructs for feeding six hours before the 24 hours' examination. If the greater part of this food is still in the stomach after this period, this observation is repeated, and, if confirmed, is regarded as evidence of obstruction. But one cannot be too careful to guard against too dogmatic an opinion in these slight cases, as there are many sources of reflex pyloric obstruction, and it is as likely as not that there is as yet no actual pathological change at the pylorus. When such a case is seen after due clinical examination and medical treatment one can, however, be fairly certain of the existence of a lesion, but when in doubt one waits till the patient is again

feeling worse, and then it is likely that such marked delay will be noted that there cannot be any doubt.

For those cases where there is a fairly large quantity of retained food, but not much atony, one instructs for feeding 14 hours before, but with the stomach that sags into the pelvis and contains retained food, through which the opaque food drops in "blobs," one re-examines without any further barium meal in the expectation that quite a large proportion will still be present 24 hours later.

It is seldom that more than the two examinations are necessary.

(4) *Hypersecretion cases* are treated as normal, i.e., fed five hours before the 24 hours examination, but if delay in emptying is noted, one re-examines on the lines indicated for pyloric obstruction cases. The nature of the secretion is tested by giving a dose of sodium bicarbonate and watching the effect on the air space.

(5) *Hour-glass contractions and neoplasms of the stomach.* No variation of the normal technique is necessary unless one suspects delay in emptying of either the upper or lower sacs. It is, however, of the utmost importance to confirm all such observations, and especially to make absolutely certain as to the presence or absence of a definite acute pain on deep pressure over the site of the neck of the contraction, or of any thickening or mass corresponding to the irregularity of outline.

(6) *Appendix cases* are treated as ordinary gastric cases, and no variation of procedure is employed, except that one frequently has the patient down for examination a third day and takes a plate of the right iliac fossa to see if any barium is left in the appendix.

(7) *Constipation cases* are followed through from day to day until one is satisfied as to the seat of the delay.

(8) *Neoplasms of the large intestine* are investigated in the same way as the constipation cases for a couple of days, and then a barium enema is given. If an appearance of obstruction to the enema is noted at any point this is confirmed by a further injection after the bowels have been thoroughly cleared.

Having recorded one's observations, one looks through all the notes, clinical and radiographic, and makes such further enquiry as the examination has suggested. Very frequently the appearances one has seen make it possible to put questions in such a way that the patient gives a much clearer account of his trouble, and one is able to come to a definite conclusion as to its cause.

CHAPTER IV.

DIAGNOSIS OF AFFECTIONS OF THE ŒSOPHAGUS.

Possibly none of the many striking conditions that are revealed by *x*-rays is so dramatic as the demonstration of œsophageal obstruction. A dogmatic positive or negative diagnosis is expected and is freely given by the radiographer, and it is usually accepted by the physician, for it is a generally accepted axiom that it is a case of either 'guilty' or 'not guilty,' and that if the bismuth food passes freely down the œsophagus there cannot be any obstruction. This is not the case, for 'obstruction' is a relative term and depends on three distinct factors, *i.e.*, (1) the consistency of the food in relation to (2) the degree of obstruction and (3) the power of the œsophageal peristalsis, aided by the action of gravity.

Moreover it does not necessarily follow that an obstruction will be always present. Spasmodic contractions of the œsophagus are just as frequent as in other parts of the alimentary tract. Where the mucous membrane is inflamed or ulcerated there will be a considerable spasmodic contraction that may, of itself, give rise to complete obstruction, although the underlying cause may be simply a small source of irritation. Perhaps the bismuth food allays the irritation and no obstruction is noted, whereas at a subsequent examination a hard particle may set up the irritation and produce the spasm.

The œsophagus, unlike the rest of the alimentary tract, has approximately only one function, namely to act as a high-way from the mouth to the stomach, and anything that interferes with this function causes the symptom of œsophageal obstruction, which may arise from a variety of causes. It is frequently the first and only sign of such serious conditions as new growths and aneurisms, whilst comparatively innocent lesions may produce precisely the same trouble. I am convinced that in some of these latter, the patient's life would have been saved by a gastrostomy if the operation had

not been put off too long, on the supposition that it was due to malignant disease and that it was not worth operating except to avert death from starvation.

To describe the œsophagus as a tubular organ of definite diameter is common in the books of descriptive anatomy. Like the rest of the alimentary canal it is a potential space when empty and is capable of considerable distension. Its diameter, for ordinary well masticated food, is between $\frac{1}{2}$ and $\frac{3}{4}$ of an inch in the upper part and this calibre is maintained throughout its length, but, as Professor Wingate Todd pointed out to me, in its lower third it is not only capable of dilatation but is actually often distended even in normal healthy subjects when the food is eaten too hastily. In other words the lower third of the œsophagus is an ampulla for the temporary storage of rapidly swallowed food where it may lie for a few moments until the narrow cardiac orifice has allowed the food to pass into the stomach. This storage action in the lower œsophagus is best seen when the stomach is hypertonic, its contraction offering a definite resistance to the entry of food into the stomach, and in such cases one frequently sees this "back pressure" dilatation of the ampulla. Occasionally in cases of hour-glass contraction of the stomach with very small upper sacs, this back pressure is so marked that all the symptoms suggest œsophageal obstruction, and in one such case the actual condition was at first missed and only discovered by accident on giving more food in order to demonstrate the passage of the œsophagus to a friend. In another such case I saw the small upper sac fill up and the patient went on eating without any difficulty until the whole œsophagus was distended up to the level of the fourth dorsal vertebra—the patient had acquired the habit of using the œsophageal ampulla to supplement the capacity of a very small upper sac of a cicatricial hour-glass stomach. Fig. 6 is only a slight exaggeration of the normal ampulla of the œsophagus.

The methods of investigation available and their limitations.

Of the bougie it is difficult to write with patience. It is an act of crude barbarity to pass such an instrument for diag-

nostic purposes into a tube whose walls may be the seat of simple or malignant ulceration or even eroded by an aneurism, if other less dangerous methods are available. The bougie is a most useful surgical instrument, and in a large proportion of cases it is the right instrument to employ for surgical purposes, but for diagnosis there is no other such barbaric relic in the whole of medicine or surgery. Plummer's method* of passing a bougie, threaded on a swallowed string, seems to be the only safe way of employing this instrument.

The œsophagoscope, on the other hand, reveals the whole of the track down which it travels but that is all. It is blind to conditions around the œsophagus and may be passed, all unsuspectingly, within a fraction of an aneurism or growth that by its pressure is causing difficulty in swallowing.

The *x*-ray method shows the shadow of the food in the œsophagus. It does not show the œsophagus itself, but it reveals the presence of aneurisms and large new growths, while from the shape and behaviour of the food shadow much may be learned as to the nature of the lesion that gives rise to the symptom. Moreover, it has two very great advantages, it is entirely free from danger and it involves no distressing manipulative procedure.

A proposition lately made by Bassler† of New York should be briefly mentioned. He makes use of a special sound with a rubber bulb at its end which, after being introduced into the stomach, is distended and pulled into the cardiac orifice which is thus occluded. In this way it is possible to distend the œsophagus, and he believes that pathological changes within the tube will thus be recognized in the very early stages. It certainly sounds a procedure that is well worth trying.

THE CAUSES OF ŒSOPHAGEAL OBSTRUCTION.

1. Those due to pressure from without.
2. Those due to changes in the walls themselves.
3. Foreign bodies.
4. Reflex causes.

* H. S. Plummer, *Journal of Amer. Med. Assoc.*, August, 1908, and June, 1910; J. S. Mayer, *ibid*, October, 1910.

† *Journal of the Amer. Med. Assoc.*, ix, No. 17, April 26, 1913.

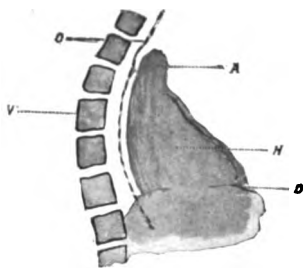


Fig. 1.

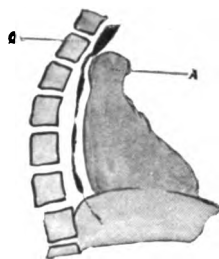


Fig. 2.

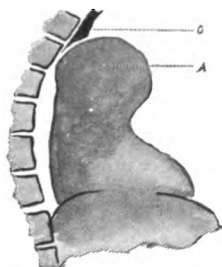


Fig. 3.

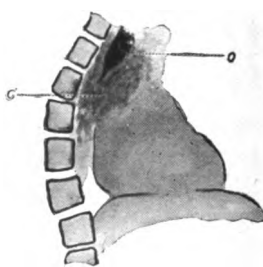


Fig. 4.

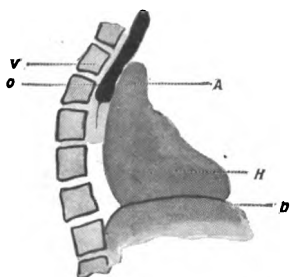


Fig. 5.

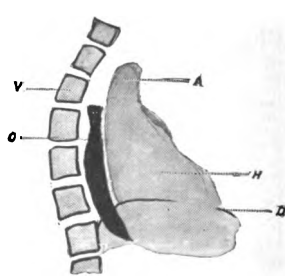


Fig. 6.

Fig. 1. Diagrammatic representation of the semi-lateral view of the posterior mediastinum with the normal course of the œsophagus indicated by the dotted line. (A. Aorta. H. Heart. D. Diaphragm. O. Œsophagus. V. Vertebral column.)

Fig. 2. Slight dilatation of aortic arch with delay of bismuth above it. No real obstruction.

Fig. 3. Large aneurism of aortic arch with œsophageal obstruction.

Fig. 4. Growth in the posterior mediastinum surrounding and obstructing the œsophagus.

Figs. 5 and 6 represent obstruction behind the aortic arch and at the cardiac orifice respectively.

- (1) *Due to external pressure.* (Cœsophageal compression.)
- (a) Aneurism.
 - (b) New growth in the neck, mediastinum, or lungs.
 - (c) Enlarged glands.
 - (d) Spinal abscess and new growths arising from the vertebral column.
 - (e) Bronchocele.

The diagnosis of these conditions does not fall within the scope of this thesis and is therefore omitted.

- (2) *Due to causes on the walls themselves.*
- (a) New growths.
 - (b) Ulceration, with spasmodic contraction.
 - (c) Cicatrization, following ulceration from caustics and acids, syphilis, etc.

It is seldom that these cases can be separated from one another. The growths are usually too small to cause any distinct shadow, while cicatrization, ulceration, and spasmodic contractions give exactly similar appearances. Belladonna is of some value in relaxing the spasmodic element in these cases I find, but it is not sufficiently reliable in causing relaxation of spasm to be of much value in diagnosis.

In this group of causes there are nearly always two factors to consider, *i.e.*, the organic and the spasmodic, and I cannot too strongly insist on the importance of this latter element which is often responsible for almost the whole of the symptomatic disturbance.

The œsophagus, like the rest of the alimentary tract, is highly sensitive, and a small abrasion or ulceration may set up a spasm of such severity and persistence that complete obstruction may result. The severity of the spasm appears to depend, not on the size of the ulcer, but upon its irritability, for I examined a case on the day following operation for removal of an eroded penny that had been lodged in the œsophagus for four months, and found only the slightest delay with quite thick food, whereas in another case, which was examined by the œsophagoscope, I found complete obstruction with dilatation although only a very small ulcer was seen. I believe that simple, or peptic ulceration of the

œsophagus is of more frequent occurrence than is commonly taught.*

It is in this type of case that the stage of dilatation with leakage or temporary recovery is sometimes seen, and a certain number of cases of simple ulceration are met with in

* The element of spasm was most beautifully demonstrated in one case. A girl of 18 complained of intermittent difficulty in swallowing and, at the first examination, no abnormality was seen to account for the trouble. Clinically, she was of neurotic type, and it was thought that the trouble was a neurosis, although she gave a history of having brought up some blood on one occasion. After careful questioning I gave her some crumbled toast and made her swallow it rapidly, and she at once said that this had produced the pain. On giving a mouthful of bismuth food it was found that there was now complete obstruction at the level of the sixth dorsal vertebra. A diagnosis of a simple peptic ulcer was made, and the œsophagoscope revealed an ulcer about a quarter of an inch in diameter, at the site indicated. It was treated locally through the œsophagoscope and also by medical measures, and eventually passed on to me to attempt ionisation. To do this was not possible through the metal œsophagoscope, and it was decided to attempt to make use of the spasm to get the bare zinc in direct contact with the ulcer. An olivary pointed and bobbin-shaped electrode was made and attached to a wire. This was all insulated except the neck of the electrode which was left bare. This electrode was easily passed into the stomach encountering no obstruction and then the patient swallowed the crumbled toast. When she told us that the pain had come on, the electrode was pulled up until the resistance of the obstruction was felt. A little extra pull brought the upper half of the bobbin through the spasmodic contraction and the neck of the electrode was felt to be firmly gripped, so that, without force, it could not be pushed either up or down. The ionisation was then attempted, but the spasm relaxed and the bougie was felt (and seen) to slip. (The procedure was carried out with the patient standing on the x-ray examination stand.) Another and deeper-necked electrode was made and the procedure was quite successfully carried out, the actual ulcerated area being marked on the neck of the electrode by the electrolysis that had taken place through this part of the metal. A complete, though temporary, cure was effected, and the patient had no further trouble of any sort for six months. The symptoms recurred, and on her return to hospital it was found that she had some septic foci about the teeth. These were attended to, but, as the symptoms persisted, another ionisation has recently been undertaken, and, so far, seems to have been satisfactory. The patient has had no recurrence for 3 months and can eat anything.

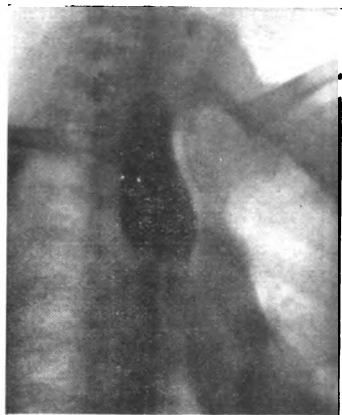


Fig. 7.

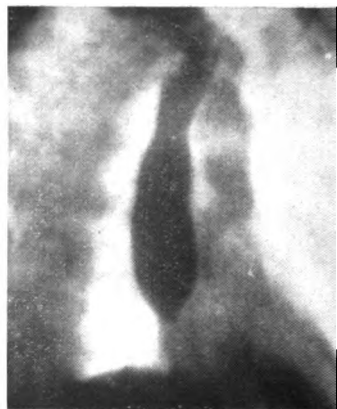


Fig. 8.



Fig. 9.



Fig. 10

Fig. 7. Radiogram of oesophageal obstruction at the level of the 6th dorsal vertebra. The cause in this case was a small peptic ulcer. The spasm that caused the obstruction was only brought on by giving dry bread. It was after this procedure that the bismuth food was given, and the radiogram indicates the complete obstruction that has resulted (See note, p. 30.)

Fig. 8. Typical oesophageal obstruction at level of 8th dorsal vertebra. It is probably intrinsic. Note the clear mediastinum around the site of the obstruction.

Fig. 9. Obstruction at cardiac orifice.—Note the indented outline of the lower end of the opaque shadow.

Fig. 10. Same case as Fig. 9, but 10 minutes later, showing how the apparent inroads of a growth were, in reality, due to retained ordinary food that has gradually been displaced and has now risen above the opaque food.

which the passing of a bougie may so stretch the base of the ulcer that healing and complete relief of all symptoms may follow, as I have seen on two occasions, one of the patients being a man of over seventy.

The use of the bougie should never be attempted unless the bismuth shadow shows a definite funnel-shape at its lower end; a bougie may wander in an amazing fashion far away from the opening into the passage. Force must never be used, and even with the gentlest manipulation, a round-nosed bougie may pass into an ulcer, and down between the mucous and muscular coats, giving a sense of absence of obstruction that, in one of the cases I examined, led to the death of the patient from direct septic extension to the lungs.

Several cases have been recorded in which new growths have perforated the walls, and bismuth has been seen passing into the pleural cavity and even into the lungs themselves.

(3) *Foreign bodies in the œsophagus.*

Most foreign bodies that are found in the œsophagus are opaque to the x-rays, and the bismuth method of examination is unnecessary for their detection. Plum-stones, fish-bones, and certain kinds of tooth-plates, however, throw no shadow that can be distinguished, and in a few of these cases I have been able to demonstrate their presence by observing the behaviour of the bismuth food as it found its way past them. In some cases the stream was divided, in others portions of bismuth were left adherent to, or in pockets about, the foreign body. On the whole, however, the detection of these translucent bodies has not been satisfactory.

(4) *Reflex causes.*

- (a) New growths and inflammatory lesions of the larynx and in the neck.
- (b) Ulceration and new growths of the cardiac end of the stomach.
- (c) Neurotic and hysterical.

(a) New growths and inflammatory lesions of the larynx and in the neck are readily diagnosed by other methods, and it is very exceptional that the x-ray examination is of any

value except in demonstrating that, although the food enters the pharynx, it does not get into the œsophagus.

(b) Ulceration and new growths of the cardiac end of the stomach, when situated close to the cardiac orifice may give rise to very marked obstruction (figs. 8 and 11). In one case a healed ulcer, two inches from the orifice, was the only pathological condition noted post-mortem in a case where the patient showed all the signs of extreme dilatation of the œsophagus. This had led to such weakness that the patient died from the shock of the operation of gastrostomy. In another case, where marked dilatation had taken place and no food appeared to enter the stomach, advanced carcinoma was found, but it did not involve the orifice which appeared to be quite patent and normal in all respects. In both these cases, although dilatation of the œsophagus had occurred, the post-mortem reports state that the œsophagus was not dilated.

(c) *Neurotic and hysterical.* I have seen no cases in which this diagnosis was confirmed, but neurotic patients frequently gave trouble by stating that they could not swallow. Persuasion and distraction of the attention, however, usually overcame this difficulty and demonstrated the nature of the case. Cardiospasm, spasmodic closure of the cardiac orifice, should perhaps be regarded as a neurosis, but in the only two cases of this lesion that have come to the post-mortem room there was definite evidence of a gastric lesion to account for the trouble, which was doubtless of a reflex nature in these cases.

Paralysis of the œsophagus is not often met with; I have seen but three cases, and in each of them it looked as if the act of deglutition was the only force at work. The food entered the œsophagus by the forceful act of deglutition and remained there, gradually sliding down in the course of many minutes with the help of drinks of water.* (Fig. 14.)

* In the first of these cases it was evident that the patient had to push each mouthful down the œsophagus by sheer forceful deglutition as far as the level of the clavicle. There was evidently no obstruction beyond this point, the food passed down quite freely and easily and not a trace was left adhering to the walls, while in the upper third traces of the food remained for a long time. It was quite clear there was paralysis in the upper third, while the lower two-

THE STAGES OF ŒSOPHAGEAL OBSTRUCTION.

Whatever the cause the result is the same and there are three definite stages, not only in the *x*-ray appearances, but also in the clinical history of the cases.

Stage 1. Difficulty in swallowing. (Most marked in upper part of the œsophagus.)

This is the stage of difficulty in swallowing. The patient has not lost weight but gives a history of some difficulty in swallowing, especially when he tries to eat his food fast and without mastication. Usually there is little or no pain, provided he masticates carefully and eats slowly, but he often states that he has to force each mouthful down separately.

On examination with the ordinary type of bismuth porridge it is probable that, at the most, a little delay is noted at one particular part. If, on the other hand, we make up our bread and milk with lumps in it and make the patient eat it quickly, we may find that there is definite delay at this same point, and occasionally one sees a violent peristaltic wave squeeze the food almost into a ball and force it through the obstruction.

The œsophagus is called upon to do an excessive amount of work, consequently hypertrophy occurs—it is the stage of compensatory hypertrophy, and for the time being compensation is established.

Stage 2. Pain after swallowing. (Not so well-marked in the lower as in the upper parts of the œsophagus.)

This is the stage of painful deglutition and the patient has begun to lose flesh. He states that he cannot swallow solids at all and that even gruel sometimes regurgitates into his mouth but he never actually vomits. The pain *after* swallowing of food is the main feature, and it is his dread of eating,

thirds were quite normal. On making enquiry from Professor Elliot Smith, I found that he had just traced out a dual nerve supply arising from two separate nuclei in the medulla. The same observation had been made by Van Gehuchten and Molhant six months previously ("Le Nevraxe," June 15, 1912, p. 55).

far more than the actual obstruction, that leads to the wasting, for although the food he eats is eventually forced through, yet the pain is such that he prefers starvation.

On examination there is definite delay at the point of obstruction; there is seldom any mistake in the diagnosis unless some such thin mixture as milk and bismuth is used, which may pass through unobstructed. The food is held up, it cannot pass on, and the œsophageal walls bring all their peristaltic power to bear on the obstruction, and as the powerful waves move downwards, the food, being unable to pass through the obstruction, escapes upwards in a narrow stream through the descending contraction. When one sees the picture one is not surprised that it is the stage in which pain is the marked feature—it is sometimes so marked that it suggests a life and death struggle. The waves are not a continuous succession of contractions; it is a case of one great effort followed by a period of comparative rest while the muscle braces itself up for another powerful contraction. This curious intermittent feature of the contractions is, I believe, characteristic of failing compensation, not only in the œsophagus, but also in the stomach, in certain stages of pyloric obstruction.

In this stage the compensation is failing and hypertrophy is about to give place to dilatation.

Stage 3. Dilatation.

(Naturally, in cases where the obstruction is high up and the œsophagus has no room to dilate, this stage is not so well marked as in the lower two-thirds of its course.)

Clinically, it is the stage of starvation and the patient is rapidly losing flesh, but the pain and difficulty in and after swallowing are comparatively slight, so that the patient feels and often becomes better for a time. There is no longer any actual difficulty in making the food pass down, but sooner or later it is brought up again—the lower down the obstruction and the greater the degree of dilatation the longer will the food be retained, so much so that in marked cases a diagnosis of pyloric obstruction is not at all infrequently made. In one



Fig. 11.

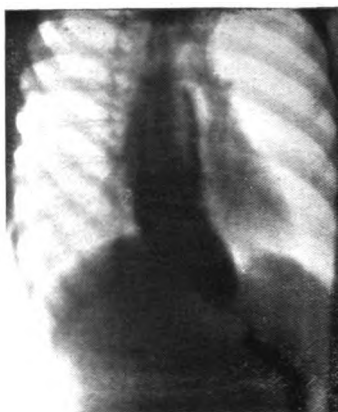


Fig. 12.

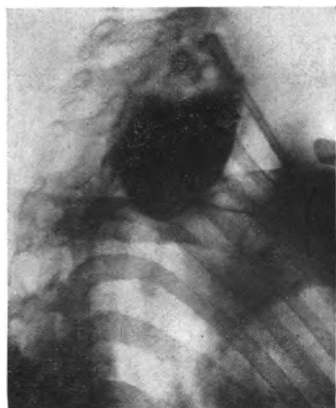


Fig. 13.

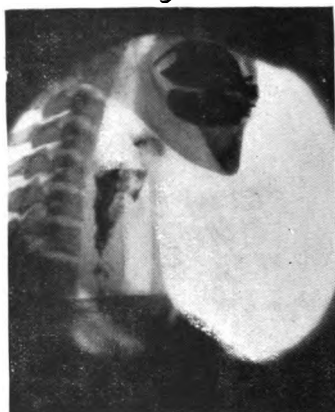


Fig. 14.

Fig. 11. Marked obstruction at the cardiac orifice showing the opaque food displacing some retained food at the lower end. The "leakage" from this case was very slow and the patient had learned to use the œsophagus for storage purposes. He suffered no discomfort from the large quantity shown in the figure.

Fig. 12. Cardiospasm in a boy aged 15. The onset was without warning and absolutely sudden when eating some food on his way home from school. Since then he had vomited almost all the food he had taken and the condition had persisted without intermission for 6 years—he was 9 years old when the trouble began—and he is now extremely emaciated. Six hours after the food had been given the shadow was still in the œsophagus, in spite of vomiting which apparently removed only the excess that the œsophagus would not retain. The food seen passing into the stomach was apparently all the opaque meal that passed through, i.e., it passed through before the spasm came into operation. (Case 2018.)

Fig. 13. True œsophageal pouch.

Fig. 14. Paralysis of upper third of œsophagus. (p. 32.)

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of these cases quite considerable quantities of bismuth were found in the dilated pouch that extended above the diaphragm two days after it had been given, and the patient gave a history of having recognised in his vomitus, food that he had taken some days previously. Indeed, in a few cases, it is absolutely impossible to recognise whether the trouble is œsophageal or gastric, for the food returned from such a pouch has the same acid smell as gastric vomit, and fermentation takes place just as readily in a dilated œsophagus as it does in the stomach in pyloric obstruction. Moreover, the stomach tube may give most misleading information, for instead of stopping at the obstruction, it will more readily pass into the pouch, whose thin walls give little sense of resistance. I have seen a tongue, $1\frac{1}{2}$ inches long, pushed out from a sac over the dome of the diaphragm by the sheer weight of the tube in a fraction of a second, in a case where an entry in the notes stated that bougies had been passed into the stomach. On more than one occasion I have found complete obstruction in cases diagnosed as carcinoma of the stomach, on the evidence of the chemical examination of a test meal that could not possibly have been in the stomach.

It would be expected that this state of affairs could not last long, but when the cause is in part spasmodic, the complete relaxation of the tone that allows the dilatation also relaxes the spasm, and this is often the most important element in the obstruction. When the cause is not rapidly progressive, a cycle may be established reverting between the second and third stages. Such cases are sometimes due to simple or peptic ulceration, which I believe to be of more frequent occurrence than is commonly supposed. The ulcer causes a spasmodic obstruction that follows through the usual three stages till dilatation is established. After a time the œsophagus is too worn out to keep up its spasmodic contraction in spite of the source of irritation, and relaxation takes place so that food passes through. Soon the muscle recovers its power and contracts on the ulcerated surface and again the spasm is produced. In the larger number of cases however, a compromise is effected and there is no definite cycle, a more or less permanent condition of dilatation with leakage being

the result, so that the patient lives in comparative comfort but in a state of semi-starvation.

On examination, no matter what the consistency of the food, it simply flows into the œsophagus and lies in the dilated sac, for there are no peristaltic waves of sufficient force to disturb it. If the sac is called upon to hold more than a certain quantity, either the patient must vomit or the sac must dilate still further. Sometimes dilatation is carried to an extreme, especially in the lower part of the œsophagus. The pouch distends along the line of least resistance which is by displacement of the lung and burrows forward over the dome of the diaphragm, occasionally even displacing the heart to some extent.

Pathologically, it is the stage where compensation has failed. The fight in which the musculature has called up all its reserves has ended in defeat. The hypertrophied wall is dilated, thinned out, and incapable of effective contraction. One would expect that at the post-mortem we should find this state of affairs as in life, but it is very seldom that it is recorded in the post-mortem notes. The only explanation I can suggest is, that although the œsophagus has been so distorted during life, in death the same conditions do not exist. Probably rigor mortis and the contraction of the elastic elements of the muscle restore the œsophagus to a semblance of the normal.

Like all involuntary muscles the œsophagus has a wonderful power of recovery, and even a grossly dilated œsophagus may completely recover its activity and tone in a very short time if rest can be secured, *i.e.*, if the obstruction can be reduced or feeding carried on by some other method. In one patient, a woman, in whom the œsophageal shadow was noted as two inches in diameter, the obstruction yielded of its own accord, and when I saw her a week later it was impossible, even with solid food, to note any abnormality.

When Cardiospasm is met with, the œsophagus is usually in a dilated condition. The appearance is of an obstruction at the cardiac orifice, and it differs in no radiographic point from the obstruction due to other causes. The history of the case will frequently extend over years, and it is often stated

that the passage of a large bougie has invariably given relief for some weeks or months. Plummer* has collected many cases of this condition and has treated them by dilatation with a bougie with great success. He has modified Mixter's suggestion of allowing the patient to swallow a silk thread which finds its way through the obstruction and eventually into the small intestine. Considerable traction can be made on the free end and the thread is used to guide the bougie through the obstruction. Olivary-nosed bougies are employed with holes through which the thread passes. In obstinate cases a dilator, an expansile bag, can be placed in position with this simple guide and the orifice stretched as much as necessary.†

DISPLACEMENTS OF THE ŒSOPHAGUS.

In the upper third of its course the œsophagus is comparatively firmly fixed, but in the lower two-thirds it lies more

* H. S. Plummer, *Journ. of Amer. Med. Assoc.*, June, 1910.

† A most remarkable case of cardiospasm was brought down to me recently by Professor Murray. The man, a well nourished coachman, stated that nine years before, while eating his tea, the food had suddenly been returned, although he had never previously had any trouble. For the whole of the nine years he had suffered from this obstruction without any intermission. On examination we found the œsophagus distended with air and a mouthful of opaque food fell, as if through space, from the pharynx to the cardiac orifice. The patient took half a pint of food without any difficulty and the picture was that of a generally dilated œsophagus about 3 inches in diameter, the bismuth food extending up to about the level of the fifth dorsal vertebra as a wide column. Above this the œsophagus was distended with air. The patient stated that he had found out that, when he had filled his œsophagus in this way, he could make the whole of the food pass into the stomach, but that it was very painful. We watched the act on two occasions, and it was evidently a very great effort on his part and took him several minutes to accomplish. There was no thin stream of food passing through at any time and, to our astonishment, the whole mass passed down into the stomach without any narrowing whatever—it was simply moved *en masse* from the œsophagus to the stomach. We are entirely at a loss to interpret this extraordinary muscular effort. The act of swallowing large quantities of air into the œsophagus and thus increasing the pressure seemed to be the *vis a tergo*, but why the whole of the food (and air also) should pass bodily into the stomach in this way we cannot understand.

or less free in areolar tissue. Various tumours cause more or less displacement; those in the region of the neck and upper thorax rapidly cause symptoms of pressure. But in the lower part of its course the œsophagus is more likely to be displaced, and one has seen a large spinal abscess producing gross displacements of the œsophagus and yet giving rise to no symptoms.

At the Seventeenth International Congress of Medicine, Dr. G. A. Pirie of Dundee recorded two very interesting cases of displacements; in one of them the œsophagus apparently wandered away into the right lung, but unfortunately its course could not be traced all the way to the stomach. The condition was of 25 years' standing and was said to have been due to ulceration.

CHAPTER V.

THE NORMAL STOMACH.

The anatomy of the soft parts, as seen in the dissecting room, is not necessarily a true index to the conditions of life. Although the stomach is more or less retort-shaped after death, it is a very different organ as it fulfils its functions in life and with the patient in the upright position. Like the rest of the alimentary canal it is a tubular organ, more or less J-shaped, and placed almost entirely to the left of the middle line. The lowest part reaches to about the level of the umbilicus,* and the pylorus is perhaps an inch higher and slightly to the right of the middle line. The actual direction of the pyloric canal differs in individuals to a considerable extent, but on the whole its direction is upwards, backwards, and to the right. These data are approximate only, as, in practice, it is found that even in patients who do not know what indigestion is, the shape of the shadow differs widely. Generally speaking, this is due to alterations in the tonic action, and therefore it is essential that this property of the gastric muscle should be grasped before any attempt is made to detect pathological changes.

There has been considerable controversy at various times regarding the normal stomach as shown by radiographic methods. This is small wonder when we compare the older anatomical diagrams of stomachs in comparison with *x-ray* appearances. It is difficult for those who have been brought up to think in terms of descriptive anatomy, to alter their conceptions to the reality of a living, sensitive organ that can contract and relax to an extraordinary extent.

In the routine examination of gastric cases I generally examine the stomach on two successive days and frequently find very marked changes in the appearances. These are

* The level of the umbilicus is practically the same as that of the iliac crests, so that there is no need to place a coin or other mark on this point to show its position.

almost invariably accounted for by alterations in tonic action : the patient feels better or is less nervous, and one notes that the atony observed on the previous day is not so marked, or vice versa. Occasionally the differences are extreme, especially in nervous subjects. But perhaps the case that indicated more than any other the extraordinary changes that are possible in the stomach was one recounted to me some years ago by Dr. Nathan Raw. A patient's abdomen was opened and the stomach was found to be a very small and extremely thick-walled organ, possibly half an inch thick, so much so that it was almost impossible to get it out of the abdomen to manipulate it. A few days later the patient died, and this small tightly contracted gastric muscle was found in the post-mortem room as a thin-walled "blotting-paper" stomach !

"The stomach of an adult is 12 inches long, 5 inches wide and its walls are about one-fifth of an inch thick . . . and has a capacity of 40 ounces,"* is the conception on which we were accustomed to base theories. That this description, from the pen of a well-known surgeon, could still appear in a leading medical journal at the present date shows that it is necessary to go over the various fallacies that have, in the past, rendered such a description possible. Granted that there are fallacies in every method of examination of the internal organs, I think that the evidence we obtain from the examination after an opaque meal is open to far fewer sources of error.

When examining the bismuth-filled stomach it is the shadow of the contents only that we can watch and this, I believe, indicates almost exactly the actual shape of the organ. That the heavy weight of a bismuth meal distorts a normal stomach is an objection that can be easily disproved by giving an ordinary meal of, say, bread and butter with tea, and then instructing the patient to drink half a pint of milk containing an ounce of bismuth, thoroughly stirred up into it. One can see the bismuth shadow descend like a veil as it permeates and flows around the food. The outline obtained in this way

* Clinical Lecture, 1914.



Fig. 15.

Fig. 15. Photograph of an empty normal stomach as we believe it to be during life. (Kindly lent by Dr. Stopford.) (p. 43.)

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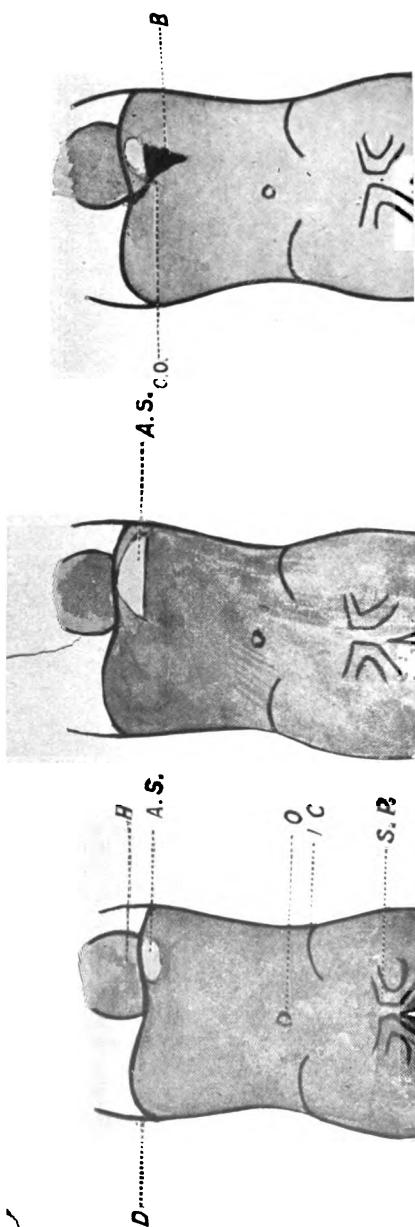


Fig. 18.

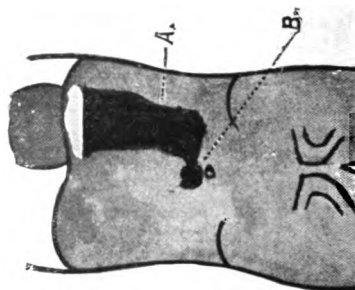


Fig. 21.

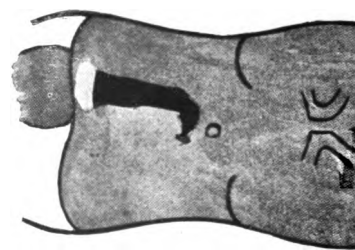


Fig. 20.

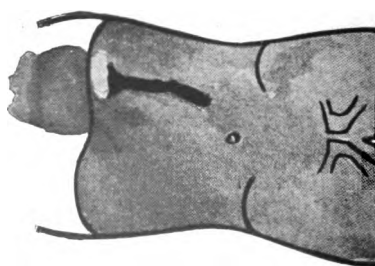


Fig. 19.

Fig. 16. Air in fundus of the empty stomach (A.S.). H., Heart. D., Diaphragm. O., Umbilicus. I.C., Iliac crest. S.P., Symphysis pubis. Fig. 17. Air in the fundus (A.S.) of a stomach that already contains fluid. Figs. 18, 19, 20, 21. Diagrams illustrate the filling of a normal stomach. B., Bismuth food. C.O., Cardiac orifice. A. and B. in Fig. 21 indicate peristaltic waves.

differs in no detail from that shown with the ordinary bismuth meal. Undoubtedly the weight of the food is of consequence, but the *normal* stomach is able to compensate for such alterations in the weight of a meal and there is no deformity as the result. When the stomach tends to be atonic it is different, and one frequently notes that the failure of tonic action is accentuated by the sheer weight of the food.

In the operating theatre there are many factors that are not those of the normal subject as he digests his food. He is lying down, *the abdomen is open*, his central nervous innervation is cut off from the abdomen to a large extent, and practically no movements are seen; the stomach is much influenced by the anæsthetic and by the shock of opening the peritoneum, and is, so to speak, fixed. Now it is easily demonstrated (p. 17) that smells and tastes that nauseate the patient give rise to immediate loss of tonic action, while in the actual process of vomiting the whole stomach contracts up in its efforts to expel the contents. My impression is, that the anæsthetic and shock "fix" the stomach at some point of the anæsthesia; the condition found by the operator is that in which the stomach happened to be when these factors came into operation. I can see no other explanation of the fact that, from the examination of the stomach with the bismuth meal, one cannot predict with any degree of accuracy whether the surgeon will find a large flaccid sac or a tightly contracted and small stomach. Indeed it is so uncertain that one even hesitates to tell a surgeon whether or not to expect a small upper sac of an hour-glass stomach, although the factor of error is halved in this case.

In the post-mortem room there are other factors which tend to produce a variety of shapes that have been described. The disease from which he suffered, the death struggles, the rigor mortis, the laying out flat (in which the natural lumbar curve of the spine disappears), all these tend to produce an organ that must be far other than as it fulfils its functions in life.

In the dissecting room, not only are all these factors present, but the body has been injected with some form of preservative, usually formalin, to which most anatomists

ascribe the varied forms that have been depicted as representing varieties of the normal stomach.*

It is highly probable therefore that the shape of the stomach as determined on the operating table, in the post-mortem and dissecting rooms, is to a very large extent a matter of chance—the wonder being that we happen to have obtained one specimen (Fig. 15) that does correspond fairly accurately with what I believe to be the normal, healthy, empty stomach. Perhaps under spinal anaesthesia more such specimens may be noted but, as yet, I have not seen any.

Like the rest of the alimentary tract, the stomach is a tubular organ, the fundus being a dilatation of the upper part. It is a great mistake to attempt to describe it in terms of descriptive anatomy and to give definite sizes and shapes, for it is a living organ, one that is perhaps more sensitive than any other in the body to both internal and external influences. For instance there are few students who have not experienced such gross changes as are indicated by the sickly sinking sensation in the abdomen while waiting for a *viva voce* examination or for the start of a race. The psychological condition is something akin to fear and produces an inhibition of the central nervous control that should regulate the tonic action of the stomach, the result being relaxation of tonic action and consequent sagging down of the greater curvature with dropping of the stomach contents to a lower level. And this is the cause of the unpleasant sensation.

The normal stomach may apparently vary within wide limits and yet be normal—the difference being accounted for by the degree of tonic action present. How sensitive is the control of this mechanism of the gastric muscles may be judged by the fact that one has seen an apparently normal stomach suddenly drop a couple of inches or more for no other reason than that a door has been slammed and has startled the patient. For this reason it is difficult to obtain a clear conception of what is actually the normal, and it is

* e.g., Patterson's diagrams, *Lancet*, June 13, 1913.

only by a study of a large number of cases, under a variety of conditions, that one can give an account of what one takes to be the normal healthy stomach.

THE FILLING OF THE STOMACH WITH FOOD.

The ordinary anatomical figures give but a poor idea of the normal empty stomach, as has already been stated. In fact, it is extremely difficult, having been brought up on descriptive anatomy, to picture the stomach as a living, sensitive organ that is capable of accommodating itself to the extremes of hunger and satiety. Dr. Stopford, of the Anatomical Department of the Manchester University, has kindly given me the use of a photograph from a specimen that he obtained. We believe it is the closest approach to the normal empty stomach that has yet been figured.* (Fig. 15.) It is the picture of a mass of muscle in just that state of tonic contraction which we believe to be normal in the healthy stomach when it is empty.†

The laws of physics obtain just as certainly in the stomach as elsewhere, and when the first mouthful of opaque food passes through the cardiac orifice into such an organ, it has to find room for itself in the potential space. Therefore a single mouthful will usually lodge for a minute or two as a V-shadow just below the cardiac orifice (Fig. 18), the fundus, filled with air, showing above it as a light half-circle—the “magenblase.” After a time, or on giving another few mouthfuls, the potential space is canalised and shows the gastric cavity as a straight line from the cardiac orifice to the lowest point, about the level of the umbilicus (Fig. 19). Here there is often a pause for a short time before the pyloric canal is filled: gravity ceases to help in the canalising of the potential space at this point, and hence the progress of the

* *Lancet*, June 13, 1913.

† Dr. Forssell's book on the musculature of the stomach has appeared since going to press, and those who are interested in the subject are strongly advised to refer to this excellent and instructive volume. (Archiv und Atlas der Normalen und pathologischen Anatomie. Über die Beziehung der Röntgenbilder des Menschlichen Magens zu seinem Autatomischen Bau. Lucas Gräfe & Sillen, Hamburg, 1913.)

shadow again halts for a short time until the intrinsic forces in the stomach itself cause the distal end to be filled by the food (Fig. 20). This pyloric portion turns off to the right, ascends, and passes backwards somewhat, so that when a small quantity of food has completely canalised the whole potential space, the shadow is roughly J-shaped.

It must be distinctly understood that these pauses are due to purely physical causes: they are not in any way dependent on specially developed muscular bands and, when once these physical forces have ceased to act, the stomach is one single cavity from the fundus to the pylorus, except when the peristaltic waves happen to segment it. Spasmodic bilocations are met with of course (p. 74), but they are quite abnormal even though they may be transient and no trace of them is seen on the operating table.

The actual shape is of little practical importance. I prefer to call it J-shaped. Holz knecht likened it to a cow's horn, while others have called it fish-hook shaped—slight differences in the pressure from fæces or air in the splenic flexure and other parts of the colon will produce various alterations such as these names suggest.*

As more food is given it is seen that the increased capacity is obtained by a widening out of the tube along its whole length (Figs. 20 and 21). The lower border of the greater curvature remains at about the level of the umbilicus (the level of the iliac crests), and the upper border is approximately on the level of the cardiac orifice. For moderate quantities (up to, say, half a pint) the whole of the increased capacity is obtained by the widening out of the tube; but when larger quantities are given there is also some slight increase of the vertical depth.

The fundus is behind the left lobe of the liver while the

* The description of the normal stomach is purposely vague—it is as fruitless to apply the precision of descriptive anatomy to such an organ as it would be to give exact figures and shapes for an india-rubber balloon. Its capacity varies from zero to many pints, while its shape is determined not only by its tonic action and the quantity of food it contains, but also by the pressure of the other intra-abdominal organs, particularly by the presence or absence of gas in the splenic flexure.

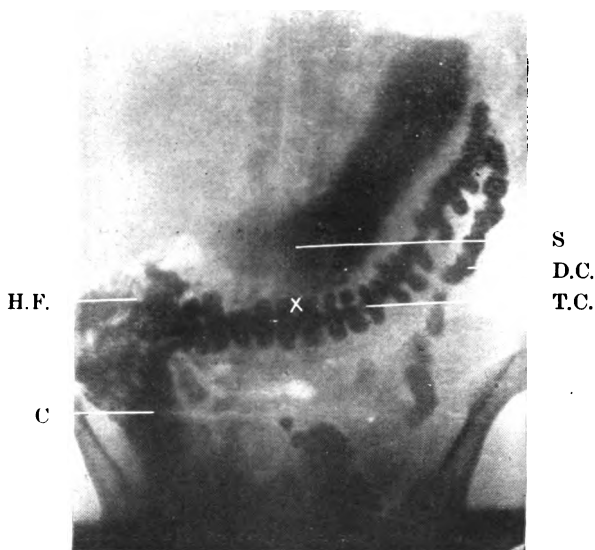


Fig. 22.



Fig. 23.

Fig. 22. Normal stomach of the J shape (S). Pyloric portion not canalised. Colon filled with the food 24 hours before. (D.C. descending Colon, T.C. Transverse Colon.) (H.F.) Hepatic flexure.

Fig. 23. Normal stomach of J shape but much displaced by large quantities of air in the colon.

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lower part comes close up under the abdominal wall, *i.e.*, the long axis inclines from behind forwards. This angle of inclination is not constant, it depends to a large extent, if not entirely, on the lumbar curvature of the vertebræ. It is interesting to note that in dogs the long axis is vertical, *i.e.*, at right angles to the vertebral column, and the whole organ is under cover of the ribs.

Of course the fundus moves with the diaphragm in respiration, but the lower border does not move at all in the healthy normal stomach, *i.e.*, there is a compensatory mechanism, a "concertina" like action, that automatically keeps the lower part of the stomach at rest; even with forced respiration there is little movement. I believe that this "concertina" action is of considerable diagnostic importance in the gauging of the degree of tonic action that is present. It is a function that is dependent on, and probably closely allied to, tonic action, and any deficiency or over-efficiency of tonic action will interfere with it. The result is that when there is slight deficiency of tonic action one can see that the lower border moves with respiration to some extent. Further relaxation interferes still more, and the lower border of the stomach may have an excursion almost equal to that of the diaphragm. But in extreme atony the greater curvature of the stomach rests on the contents of the pelvis and is not disturbed by even forced respiration. On the other hand, when tonic action is more marked than usual, the stomach is contracted up and there is not the same scope for this compensatory mechanism. Consequently some movement is seen, and sometimes, when one meets with a very hypertonic stomach, contracted up to perhaps half its normal size, there may be no trace of this "concertina" action to be seen.

I find the observation of this "concertina" mechanism of the stomach is of considerable assistance as an indication of the tonic action that is present.

Manipulation, "radioscopic palpation" of the normal stomach shows that the organ is quite freely movable. In suitable cases, when the abdominal wall can be well relaxed, the lowest part can be pressed upwards a couple of inches :

the lower two-thirds of the organ can be displaced laterally about one and a half inches towards the liver, and perhaps an inch towards the spleen. Even in muscular men considerable movement can be obtained. The pyloric portion is more fixed, but a certain amount of movement can nearly always be produced. The upper third of the stomach is under cover of the ribs and cannot be reached by palpation.

Practice is needed in palpation in the upright position but, owing to the action of gravity on the organs themselves and on their contents, it gives so much more information as regards the stomach than palpation in the horizontal position, that everyone should avail himself of opportunities to become accustomed to this most useful assistance in diagnosis.

In certain cases one finds that, by pressure, one can make a certain quantity of food pass from the stomach into the duodenum. If the hand is held over the left side of the patient's abdomen and a sudden push applied when a wave of peristalsis is about an inch from the pylorus, it is very often possible to push quite a considerable quantity through the sphincter, especially in those cases that show the symptom-complex of duodenal irritation.

In a paper* before the Electro-therapeutic section of the Royal Society of Medicine, I showed that the contents are not churned up but take up their positions according to their specific gravities, except in the pars pylorica when the peristalsis is active; consequently air always occupies the fundus, and the heavier food gravitates to the lowest part. Hence the bismuth food outlines the lowest border in all cases.

If the stomach is empty, the air appears as an oval clear space (Fig. 16), while, if there is a quantity of food in the stomach, the space represents the arc of a circle, its lower border being a straight line which can be demonstrated as fluid by watching the ripples on its surface on shaking the patient (Fig. 17).

Working with Dr. Maude McNaughton, we found that in infants the shape of the organ is much more globular, and during the first year of life is comparatively spherical, but as the child assumes the upright position the stomach elongates.

* "Proc. Roy. Soc. Med.," Feb., 1909.

It is not, however, till near the age of puberty that the lower border descends to the level of the umbilicus.

This is the description of the normal stomach, but, as I have already said, it is by no means always found in persons who have never suffered from any gastric trouble. It is a fact that the stomach may present *an appearance* that is far from normal and yet fulfil its functions perfectly. The part played by tonic action or defective tonic action in the appearances seen is the key note to correct interpretation in these cases, and will therefore be considered.

TONIC ACTION.

The upper border of the contents should be about the cardiac orifice, and the increased capacity for more food should be obtained by a widening of the tube. (See Figs. 18 to 21.) Tonic action, therefore, may be defined as the constant contraction of the stomach which maintains the contents in tubular form, *i.e.*, it is an automatic contraction that counter-balances the action of gravity on the stomach contents. In the recumbent position therefore, the tonic action is not called upon to anything like the same extent, and it seems reasonable to suppose that the benefit received from rest in bed is due, in part at least, to the relief of the constant strain on this function.

A thorough understanding of this property of the muscle is invaluable in gastric radioscopy. Tone is a property of the living muscle, and is therefore not seen in the post-mortem room. It is under the control of the central nervous system, and may alter very rapidly. For instance, everyone knows the sinking feeling that accompanies nausea, sudden fear, and disgusting smells, and in these conditions I have invariably found that this sinking sensation in the abdomen is accompanied by an actual relaxation of the tone, and consequently by a drop in the level of the lower border of the stomach, while an increase in the tone accompanies the actual process of vomiting and retching. I have made many experiments on tonic action with various drugs such as valerian and asafoetida, and the loss of tone seemed to be in direct proportion to the disgust in almost every case. I have also attempted

stimulation of tonic action by such things as liqueurs and *hors d'oeuvres*, with rather less than more success. Sometimes a slightly atonic stomach would apparently be braced up by such items as appealed to the patient's senses, but, more often than not, the selected patients, with atonic stomachs, evinced no great relish for these luxuries, and there was no appreciable change in the condition of the stomach.

Loss of tone is often associated with loss of appetite, while appreciation of food tends not only to stimulate the flow of gastric juice, as we know, but also to increase the tone. For this reason it is important that, so far as possible, the patient's tastes should be considered in the matter of the character of food given. It should be as palatable as possible; the addition of sugar usually covers the chalky taste of the opaque meal sufficiently. And the food should be presented in a pleasing manner; it should be nicely served and his mind should be set at rest—in all things, the patient's comfort, both bodily and mental, should be studied. But it is essential that at least the first few mouthfuls of the food should be eaten while the patient is standing before the fluoroscopic screen, otherwise some point of great importance may be missed as the food canalises the empty stomach.

PERISTALSIS.

It should be possible to make out the peristaltic movements in all except the very stoutest of patients; the waves appear to be always present from the time the food enters till the stomach is empty. They start opposite the cardiac orifice and sweep along the greater curvature, gaining in force as they approach the pylorus. With the ordinary opaque meal each wave is like its fellows, but if there are lumps in the food there may be some inequality in the peristalsis as the result.

On the lesser curvature the waves are not seen so high up. When they actually segment the bismuth shadow some distance from the pylorus, the peristalsis is more powerful than normal. It does not necessarily happen that the same stomach exhibits exactly the same character of peristalsis on all occasions: some days the waves are stronger than others,



Fig. 24.



Fig. 25.

Fig. 24. Small hypertonic stomach—reproduced on same scale as 22 and 23.

Fig. 25. Also reproduced on same scale. A hypertonic stomach containing a fairly large meal (for a small hypertonic stomach). The colon contains the meal given the day before.

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but on the whole it will be found that they are fairly uniform in character and periodicity.

In certain cases, however, it is seen that there are periods of marked activity followed by periods of comparative, or even complete, rest. In others an occasional wave or succession of waves of far greater strength are noted. In both of these types there is a very strong suspicion that there is some pyloric obstruction, even if there is no actual delay in emptying. Delay in emptying only occurs when the compensating peristalsis begins to fail—the intermittent strength of the waves is an indication of failing compensation. In fact, in several cases the first examination has shown rapid emptying in association with very active peristalsis, whereas another examination, conducted when the patient was feeling ill, revealed a typical pyloric obstruction with marked delay in emptying.

The waves pass at about the rate of three a minute and are practically uniform in character, each wave being the same in size and strength as its predecessor. But in cases of pyloric obstruction this is altered; at first there is increased peristaltic action and this compensates for the obstruction, but later on, the stomach gets worn out, and this is evidenced by the fact that we see uneven peristalsis; one wave of great strength followed by a period of rest or a series of feeble and obviously incompetent movements while the organ is resting before again making a big effort.

The persistent absence of peristalsis in a stomach otherwise normal in appearance but containing retained food, is associated with extensive ulceration—usually malignant—involving the pyloric region and giving rise to obstruction.

Naturally, in a thinned-out atonic stomach, the peristalsis is likely to be much less powerful than in a normal stomach, but even in extreme cases, when the lowest part is almost in the pelvis, peristalsis can always be elicited by massage. In these cases it is difficult to observe the movements; the shadow of the sacrum and ilium obstruct the view.

Reverse peristalsis is seldom seen in this country and when met with indicates gross pathological changes, usually at the pylorus, but I have only seen it in ten or twelve cases.

In several cases I have noted that this feature was apparently of an intermittent nature, as if the waves were uncertain as to which direction to take. Sometimes a wave would hesitate and apparently become stationary and then proceed in the reverse direction.

In one instance (a typical cicatricial pyloric obstruction) where this hesitancy was noted, the application of a little gentle massage to the greater curvature was sufficient to restore the peristalsis to its normal direction. (See footnote p. 64.)

THE RATE OF EMPTYING.

The rate of emptying varies greatly, and is of course influenced by:—

1. The quantity of food.
2. The quality of the food.
3. The various factors in the stomach itself.

For bismuth examinations the quantity of the food is from three to fifteen ounces, and this should easily be dealt with in three or four hours under normal conditions.

The quality of the food is of importance, for we know that fatty foods tend to delay emptying, but employing a reasonable quantity of carbohydrate (bread and milk) opaque meal, it will be found that in normal healthy subjects the stomach is empty in three hours.

Apart from actual obstruction there are certain physical causes that tend to delay emptying, *e.g.*, when atony is present the food sags the greater curvature down, far below the pylorus, and there is this mechanical disability to be overcome. In many cases the atony is the effect of perhaps slight obstruction, and this mechanical factor, added to the organic lesion, is of considerable importance—even a comparatively slight obstruction in association with marked atony will result in great delay in emptying.

We can control the quantity and quality of the food, but we cannot control the motor functions of the stomach, and these vary from time to time for a variety of causes. For instance, it happens that some delay is noted when the subject is an out-patient, whereas the rest and regime of in-patient life quickly restore the jaded functions and no delay is

observed; and invariably the patient tells you how much better he is since he came into hospital. I am convinced that in many of these patients it is simply the rest in bed—giving rest to the tonic action of the stomach so that it is not persistently called upon—that produces this effect (which persists for just two or three weeks after they leave the wards and resume their daily life).

Again, the patient's symptoms come and go from time to time even while at work, and if we examine between attacks we may find a very different rate of emptying to that which we record when the actual attack is present. In other words, the early symptoms of pyloric obstruction are often dependent on spasmodic contractions.

The patient's feeling of ill-health, out-of-sorts, or depression, is sometimes dependent on atony and delayed emptying, whereas his feelings of well-being under hospital treatment, with suitable food and rest, is the effect of better tonic action and the resulting improvement in the efficiency of the motor functions of the stomach.

Swallowed septic matter from teeth seems to be a source of pyloric spasm, and I have now seen many cases of apparent pyloric obstruction cleared up completely by wholesale extractions and overhauling of bridge-work and crowns that so often cover veritable cess-pools, discharging into the alimentary canal.

There is also a type of case in which the rate of emptying is extremely rapid to start with. One sees the small intestine overloaded in a quarter of an hour or less and yet after five or six hours there may still be a considerable quantity of food left in the stomach (see ileo-pyloric reflex p. 54).

To say that because the stomach is not empty in five or six hours there must be pyloric obstruction, is therefore too sweeping a generalization. All possible sources of error must be first excluded. But when all foci of septic absorption have been excluded and rational treatment, dietetic and otherwise, has been tried without success, then a diagnosis of obstruction may be made on such comparatively slight delay as five hours, provided it is persistently noted on two or three separate occasions. But in the majority of cases

where organic obstruction exists there is seldom any doubt, practically the whole of the food given six hours before is still present, and in the marked cases the 24 hours' food is also still in the stomach. In extreme cases opaque food remains in the stomach for many days, in spite of persistent vomiting.

THE CONTROL OF THE PYLORUS.

By means of giving sodium bicarbonate and observing the evolution of carbon dioxide, the increase of size of the air space, the acidity of the gastric contents can be roughly estimated, and it is certainly not in those cases in which the evolution of gas is greatest that the pylorus opens most freely, but in the class of case that I have styled 'duodenal irritation' (p. 99). In this type of case the food is seen passing out almost at once into the duodenum and continuing to pass out rapidly—one does not see any hypersecretion and the evolution of gas is not more than in healthy people. It looks therefore as if the degree of acidity of the gastric contents is not the determining factor.

Such evidence as I have seems to prove that fatty foods tend to remain longer in the stomach than the carbohydrate and protein meals usually employed, but I have not noted any difference in this respect between the porridge and bread and milk opaque meals.

The rapid emptying in the duodenal cases is most striking. Not only does the food go through more rapidly, but also the pyloric relaxation seems to be much more complete than in the healthy subject, with the result that quite large quantities pass through at a time and are easily seen on the fluorescent screen—in contrast with the finer division and thin stream in which the food leaves the stomach in perfectly healthy subjects, which can only be detected on the fluorescent screen under the most favourable conditions. In a certain number of these I have also noted that the pylorus did not open at once and no food was seen passing through for perhaps ten minutes, but when once shadows were seen in the duodenum, the stomach began to empty rapidly as if the mere presence of food in the duodenum brought about pyloric relaxation. In every case of actual ulceration of the duodenum this rapid emptying has been noted, whether the meal was made up of

bread and milk or of porridge. Distending the duodenum does not bring about closure of the pylorus, rather the reverse, so far as I can tell from observations on a limited number of cases in which this portion of the intestine has been overloaded either artificially by giving a very large meal, or pathologically by reason of cicatrization.

These observations suggest that the control of the pylorus is regulated by some sensory mechanism in the duodenum. An irritable state of this part of the gut, whether intrinsic or reflex, tends to abnormal pyloric relaxation. In some cases this relaxation is so marked that one can press perhaps an ounce of food through at a time if the pressure is applied at the right moment, *i.e.*, when a wave is about an inch from the pylorus. In the normal it is seldom possible to see more than a mere trace pass into the duodenum by this means.

A curious feature of a certain proportion of these duodenal irritation cases is the fact that, although they commence to empty so rapidly, embarrassing the small intestine by the large quantity allowed to pass, yet, after a certain period, there comes a time when the stomach ceases to empty in this manner. In point of fact there may be actual delay in completely emptying the stomach. Sometimes this is so marked that, had one started the examination by feeding the patient six hours beforehand, one would undoubtedly have diagnosed pyloric obstruction—one of the many instances that show the importance of examining right from the start and with the stomach empty. On careful examination one finds that the overloading of the small intestine proceeds more or less regularly, and palpation shows that it is not one particular coil that is distended but that it is a general overloading of the small bowel. But in certain cases the emptying of the stomach ceases, the peristalsis quiets down, and there appears to be delay in emptying. This always seems to occur when there is a heavy collection behind the ileocaecal valve. (J. T. Case* and Hertz† have shown how this valve acts, not to prevent regurgitation from the large intestine but to control

* *American Quarterly of Röntgenology*, Nov., 1912.

† *Journal of Physiology*, Oct. 17, 1913.

the passage of the ileal contents into the large bowel.) One sees this sequence of events so often in this class of case that there can be little doubt as to the interpretation, *i.e.*, an ileo-pyloric reflex, yet one more of these alimentary canal reflexes that make the symptomatology of disease within the abdomen so very complex and elusive. It is a reflex from the last coil of the ileum back to the pylorus, a message to indicate that as much food has come to this point as can be dealt with and calling for the shutting off of the supply, the response being a quietening down of the peristalsis and a closure of the pylorus. The importance of this reflex in connection with dyspepsias due to trouble in the cæcal region is obvious.

In this type of case one sometimes finds that giving more food again starts the rapid emptying, and on more than one occasion I have at the same time noted the passing on of food from the ileum and also movements of the cæcum. Case* and Hertz† independently showed how the giving of a meal is often a signal for the passage of the contents of the ileum into the cæcum, and, if one interprets these appearances aright, it means that in those cases where food again promotes the rapid emptying, the ileo-pyloric reflex was just ceasing to act.

That such a reflex as this existed was foreshadowed by Lonquet‡ in 1902, but, so far as I know, no definite theory on the subject was advanced until Sherren§ again brought forward the theory of which we now have proof. Speaking of appendix dyspepsia he says: "It is obvious that the sole cause cannot be that the appendix is diseased, for I have shown how often and how grossly this may occur without giving rise to dyspeptic symptoms. That these occur most often with an appendix that is astonishingly innocent looking, appears to be proof that reflex interference with the action of the stomach, the result of spasm in the region of the ileo-cæcal valve in some cases, mechanical in others, or intestinal stasis, must be the cause of the symptoms.

"I believe that the part played by the diseased appendix in disease of the stomach is twofold. It may reflexly produce

* *Am. Quarterly of Röntg.*, Nov., 1912.

† *Journal of Physiolog.*, Oct. 17, 1913.

‡ *Semaine Méd.*, 1902, 185.

§ "Relation of Appendicitis to Dyspepsia," *Brit. Journal of Surgery*, vol. i, No. 3, 1914.

pyloric spasm, or may interfere with the lower ileum directly.

The control of the pylorus therefore appears to be a most complex mechanism and probably depends far more on the conditions obtaining not only in the duodenum but also in the lower alimentary tract than on the acidity of the gastric contents, the composition of the foods and the other factors that we used to consider as the controlling influences. It looks as if the duodenum is, as it were, the central office for controlling the distribution from the stomach, and is in direct communication with all the lower alimentary tract by a complex nervous mechanism of which we, as yet, know next to nothing.

THE CONTROL OF PERISTALSIS AND TONIC ACTION.

As I have pointed out,* I believe that tonic action and peristalsis are entirely separate and independent muscular functions. The evidence on which this opinion is based is, that even in the cases where atony was most marked, peristalsis was quite well seen, while in a limited number of instances (type 2 of chronic pyloric obstruction†) an exactly

* Proc. Roy. Soc. Med., Electro-Therapeutic Section, Feb., 1909, p. 9.

† In advanced cases of pyloric obstruction two definite and widely differing types are met with and will be discussed in detail later (p. 64) :—

Type 1. Is far the most common. The stomach is extremely atonic and sags down into the pelvic. Peristalsis may be quite active and yet the food may be retained for days.

Type 2. Is rare. The stomach is normal in shape, but distended with retained food, and the bismuth is seen in the stomach for 24 hours or longer. Frequently in these cases no peristalsis is seen and no movement can be evoked by massage.

* * * * *

Prof. Wingate Todd suggests that in Type 1, the atonic type, it is the long fibres that are atonic, while the circular fibres are not worn out; hence peristalsis goes on quite freely. But in type 2 the hyper-tonic type, the reverse condition obtains, *i.e.*, there is atony of the circular fibres while the long fibres are unaffected. He believes that the long fibres are responsible for the tonic action, while the circular fibres fulfil the function of peristalsis.

opposite state of affairs was found, *i.e.*, perfect tonic action with complete and persistent absence of peristalsis.

That both of these functions are influenced by the central nervous system has been demonstrated in a variety of ways. For instance, tonic action and even spasmodic contractions have been relaxed when patients have been frightened. When patients have become faint I have nearly always had warning by seeing a sudden relaxation of tonic action—an observation that has sometimes saved the patient from a nasty fall in the midst of the apparatus. If there are a number of observers present the patient tends to become nervous, and often I have failed to demonstrate excessive peristalsis and hypertonic conditions that were invariably present when I carried out the observations by myself. The influence of nauseating smells and tastes in causing relaxation of tone has already been referred to (p. 17).

In the early stages of pyloric obstruction, as we should expect, peristalsis is more marked than in health, but the phenomenon of very active peristalsis and hypertony, in association with an *abnormally patent pylorus* in cases of duodenal ulcer and duodenal irritation, suggest that these functions of the musculature are also influenced by some reflex connection with the duodenum.

In the cases referred to as type 2, of chronic pyloric obstruction, we have complete absence of peristalsis. Neither massage nor electrical stimulation seem to be of any avail in eliciting contractions. In these cases also an almost hypertonic condition persists and is quite uninfluenced by attempting to induce nausea. It is most striking that it is only in this class of case that neither of these functions can be influenced by any means I have tried, and the inference is that the nerve supply which regulates them is cut off. The actual pathological lesion found has always been of an extensive nature involving the lesser curvature in the pyloric region, and presumably this is where the nerve supply passes into the stomach from the duodenum, either directly or through sympathetic ganglia. The suggestion, therefore, is that the injury to these nerves produces a complete absence of peristalsis while tonic action is very pronounced, a condi-

tion that is in marked contrast to the usual atonic condition found in type 1 of chronic pyloric obstruction. It looks as if tonic action is an intrinsic muscular endowment, or that there are centres controlling this function in the stomach wall itself, or connecting with the vagus, and acting in a similar manner to the centres in the lumbar enlargement for the regulation of micturition. In the vast majority of chronic pyloric obstruction cases, I have found that peristalsis is present and that the stomach is atonic. In some of these the pathological conditions were precisely similar to those found in the rarer type of case. If therefore my deductions are correct, it seems probable that the production of one or other type depends on whether or not these communicating nerve fibres are interfered with by the progress of the disease.

NOTES ON CASES IN WHICH THE OPERATION REVEALED A
NORMAL STOMACH. (*Cases on pp. 143-4.*)

In the cases filed under this head are included only those in which no abnormality involving the stomach was found at the operation, with the exception of the duodenal cases, and those in which the x-ray picture of 'duodenal irritation' was present, which have been tabulated under a separate heading.

For a description of the normal stomach, see p. 39.

In many apparently normal stomachs a certain degree of spasmodic contraction of the middle of the body was noted, but in most of them the application of massage to the abdomen relaxed the spasm at once, while in a small number, notably Nos. 369 and 398, the contraction was thought to be organic in origin as it could not be relaxed by massage or the administration of belladonna. In case 398 this spasm persisted, and was so definite that at my instance the surgeon again explored and found nothing whatever to account for it; while in case 369 the simple manipulation of the stomach at the operation was sufficient to effect a perfect cure, and re-examination after the laparotomy showed no trace of the contraction previously noted.

Displacement of the lower part of the stomach was often met with. The most common cause was distension of the colon with air or fæces, and the examination 24 hours later

nearly always made this point quite clear. Adhesions and new growths were also factors in displacements, and the detection of these conditions depended, as a rule, on manipulation of the organ, preferably when the colon was also filled with bismuth. Under this heading are tabulated only those cases in which the stomach itself was not involved by the lesion.

Case 296 is curious, in that all observers detected a definite abdominal tumour near the pylorus. Radiographically I found no evidence of any abnormality, and no tumour could be found at the operation.

Slight or even marked atony was noted fairly frequently but was regarded rather as deficient physiological contraction than as a pathological condition.

CHAPTER VI.

THE PATHOLOGICAL STOMACH.

ATONY.*

Atony, the failure of the muscle to maintain the tubular form against the action of gravity, is a complication of many pathological conditions of the stomach, and the part played by atony in the resulting shadow of the bismuth meal must be appreciated before we can diagnose other lesions of which the atony is merely a complication.

In the mildest degree of atony, so frequently met with, the food is held up for a short time, and then gradually gravitates to the lowest part; there is, however, always some evidence of the tubular formation remaining, even after a prolonged period. Perhaps the actual weight of the opaque food is a factor in these cases and with ordinary food this deficiency would not occur. In marked cases the food straightway gravitates to the lowest part, and it is only the lowest border that is outlined. The picture of a really atonic stomach, as seen upon the screen, shows that the bismuth meal, instead of being held up in tubular form, sinks at once to the lowest point where it lies as in a sac. The whole bismuth meal quickly finds its level in the lowest part, forming a more or less crescentic shadow low down in the abdomen, and in extreme cases even on a level with the pubes.

In such cases it is often difficult to persuade the patient to take more than a very small quantity of the bismuth food, but if he can be prevailed upon to do so, it is found that the increased capacity is obtained by an increase in the vertical depth of the shadow. In other words the stomach does not contract upon its contents but appears to remain inert; it is a toneless sac.

The manner in which the food enters the stomach suggests

* Strictly speaking atony should be considered with the normal stomach for it is a defective physiological action rather than a pathological condition.

that the walls of the upper part are in contact, *i.e.*, it flows down in a thick stream. Sometimes, at the junction of the middle and the upper third, the stream breaks off in thick 'blobs' which drop to the lowest point, like tar falling through water, showing that there is already some food in the lower part of the stomach keeping the walls apart, and through which the bismuth food sinks.

Fallacies in the diagnosis of atony.

That the bismuth shadow only outlines the lowest part of the stomach after a time does not necessarily indicate loss of tone, for it must be remembered that the stomach itself will be secreting juice, which, being lighter than the bismuth food, will consequently rise above it. Hence, although the tubular form may be maintained, yet the upper part of the tube will throw no shadow, because it contains none of the bismuth mixture, for, as I have already said, there is no churning action in the stomach except close to the pylorus.

For the same reason an atonic condition is suggested when the stomach already contains food; the bismuth mixture rapidly sinks through the stomach contents, which cannot be seen, and hence only the lower border is outlined. The fact of a full stomach is always suggested, however, before the bismuth food is given by the shape of the air space, bounded below by the upper margin of the fluid. Also, when the bismuth food enters, it passes down more rapidly than when canalizing a passage between the collapsed walls, and in a manner that at once suggests a heavy substance falling through a lighter. Moreover, when a considerable quantity has been given, the shadow indicates that it is only the lower part of a column that we see, and further information on this point is easily obtained by pressing the stomach contents upwards.

The lowest border of the stomach is well below the umbilicus in atony, but this is no proof that the stomach is atonic, for the whole organ is displaced when the diaphragm is below its usual level. This condition—visceroptosis—is most important, as it gives rise to severe gastric symptoms in some cases.

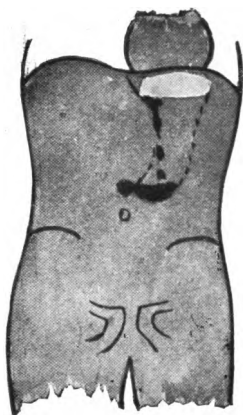


Fig. 26.

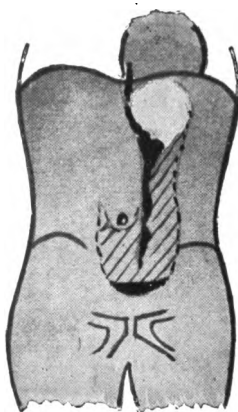


Fig. 27.

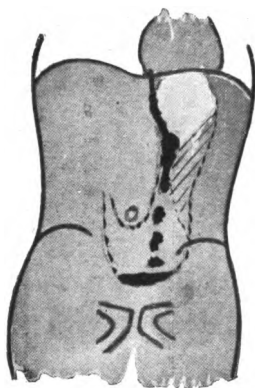


Fig. 28.

Fig. 26. Diagram of bismuth food entering a stomach that already contains food or fluid, possibly secretion. Note the "blobs" dropping through the lighter food.

Fig. 27. Diagram of food entering an empty atonic stomach. The shaded area represents the collapsed walls as I imagine them to be.

Fig. 28. Diagram of food entering an atonic stomach which already contains food. Note the "blobs" dropping through the retained food.

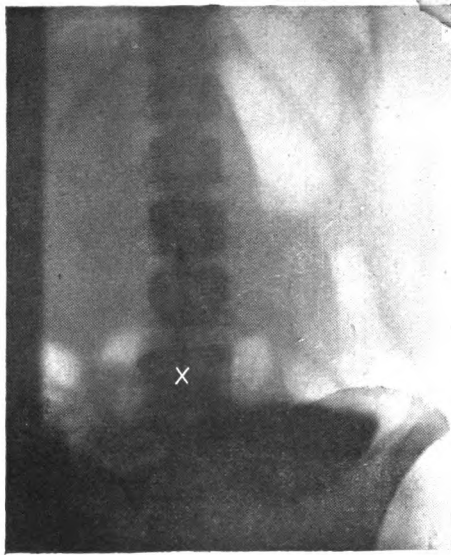


Fig. 29.

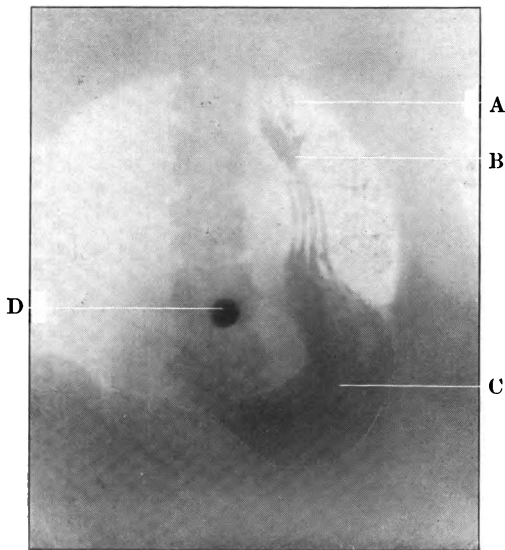


Fig. 30.

Fig. 29. Atonic stomach—the food has all dropped to the lowest part. Note the elongated air space as in the diagrams illustrating borborygmi (figs. 53). The patient exhibited the borborygmi most perfectly.

Fig. 30. Radiogram of an atonic stomach. Pressure has been applied to the abdomen so that the food has been forced upwards. The photo was taken as the food was again taking up its position in the lower part and shows the distribution of the rugæ in parallel lines coinciding with the axis of the stomach. (A) air, (B) food held up between the collapsed walls and passing down through the rugæ to the mass of food, (C), (D) umbilicus. A suspension of bismuth was used to obtain this result.

In a certain number of cases where we find the lowest border of the stomach far below the umbilicus, the tonic action appears to be quite good. This condition is known as *gastroptosis*, and it is said by Goldthwaite* that faulty posture is the chief factor in its production. Certainly it can be greatly improved by abdominal massage and exercises that tend to increase the normal lumbar concavity, as I have proved on several occasions. (Fig. 31.)

It is a condition that places the gastric peristalsis at considerable disadvantage. Since the pylorus is well above the lowest part there is a tendency towards retention of food in the stomach, which in its turn will lead to a constant strain on the tonic action, and if this gives way, the lower border falls still further with the result that there may be delay in emptying.

Moreover, the transverse colon is attached to the lower border of the stomach by the transverse mesocolon, and it follows that in these cases this portion of the large intestine is also found below the usual level. It is seldom that the ascending and descending colon have any mesentery and consequently there is a tendency to kinking of the large intestine at both the splenic and hepatic flexures, although I am not convinced that it actually occurs. As these patients appear to suffer from constipation it is probable that mechanical obstruction may play a part, and Goldthwaite believes that this condition with its resulting constipation is the cause of many diseases, such as rheumatoid arthritis. He records most encouraging cases in support of the treatment by massage and exercises. In extreme cases he advises a short-circuiting operation.

Frequently at operations the stomach which has appeared at the *x*-ray examination to be quite normal is found as a large flaccid sac, and *vice versa*. My explanation of this discrepancy is that nausea, disgust, and fear, bring about relaxation of tonic action, *i.e.*, the stomach will tend to become a large atonic sac, whereas I have always found that

* Goldthwaite, *Boston Med. and Surg. Journal*, 1904, and May, 1906.

in the act of retching or vomiting it is contracted up. In anæsthesia the gastric muscle is found in varying degrees of contraction and relaxation; one can never say for certain what the condition will be when the abdomen is opened. Usually the large atonic stomach of pyloric obstruction is found as a great flaccid sac, but the radiographically hypertonic and normal stomachs may appear, on the operating table, in any condition from tightly contracted to large flabby stomachs. The muscle is profoundly influenced by the anæsthetic and the shock of opening the peritoneum; occasional halting contractions are seen, caricatures of normal peristalsis, while the tonic contraction seems to be more or less fixed—it is a very different thing to the accommodating and automatically compensating organ that we know from radiographic studies. It seems therefore as if the gastric muscle is 'fixed' in some stage of the anæsthesia, and my suggestion is, that the condition found on the operating table is that which happened to be present when the anæsthetic acted upon it. If this occurred during a period straining, a tightly contracted stomach will be found, but if nausea happened to be present the organ will more likely be seen in a relaxed state.

VOMITING.

In the administration of opaque meals one meets with subjects who say that the food will make them sick. Most of them are women of neurotic type who have got into the habit of vomiting, and take only very small quantities of food at a time. It is seldom that they do actually vomit, and with a few words of assurance and encouragement they generally take as much food as is necessary for the examination. The stomach in these cases is nearly always of the atonic type. But there is another class of patient who says that the food will make him sick. They are chiefly men with hypertonic stomachs who cannot take food quickly (the hypertonic stomach takes time to accommodate the quantity of food), and if one goes on feeding there is a backing up of the food into the œsophagus. It is likely that some will come back unless the patient is allowed to take his own time over eating.

When once the food has been taken, however, it is comparatively seldom that vomiting occurs, but I have had a certain number of opportunities of observing the process. There is often a period of malaise and nausea, and in this stage a considerable loss of tone is noted in the stomach, the lower border dropping several inches. But when once the act of vomiting commences, not only are the diaphragm fixed and the abdominal muscles brought into play, but there is a general contracting up of the whole stomach in its efforts to expel the contents. Comparatively violent peristaltic waves are noted, and in the retching stage the contents of the stomach are churned up.

The contents of the upper part of the stomach are those that are first expelled, and when there is free secretion going on, as often happens before vomiting, it is the fluid from above the opaque food that is first returned. In the hypertonic, and less often in the normal stomach, one has seen nearly the whole of the contents brought back, but when atony is present it is very seldom that more than a small portion of the upper part of the food is returned. When atony is marked, as in old-standing pyloric obstruction cases, even the act of vomiting is incapable of contracting the stomach, and only a fraction of the contents comes back. Since the opaque food all lies in the lowest part, this is the last to be expelled; therefore it is only the stomachs of normal or hypertonic type that are capable of emptying the bismuth food more or less completely. But in the atonic type of stomach even the most violent attacks of vomiting fail to eject the opaque food, and in one patient, one of the very early cases (No. 10), I watched the food remaining in the stomach from day to day for nearly a week, in spite of persistent vomiting.

In hour-glass stomachs of the cicatricial type it is, I believe, only from the upper sac that vomiting occurs, but one was more surprised to find that in certain of the spasmodic hour-glass stomachs—where subsequent operations showed absolutely nothing to account for the hour-glass contraction—the patient vomited from the upper sac only, the discomfort and desire to vomit passing off as soon as the upper sac was relieved.

In this connection an interesting personal experience was related by Dr. Deane Butcher.* :—

"Some years ago he accidentally swallowed an irritant poison. He rushed into the surgery and hastily swallowed several tumblers of hot water containing permanganate of potash. The first vomiting was clear, pink, and unmixed with food. Only after three or four repetitions did food appear in the vomit, although a copious dinner had been taken three-quarters of an hour before. This unintentional experiment seemed to prove the occurrence of an hour-glass contraction of the stomach as a consequence of irritation."†

In several cases I have seen regurgitation of food from the small intestine into the stomach. These have always been associated with gross obstructions at the duodeno-jejunal flexure or in the jejunum. In one of the latter a man with an early carcinoma of the jejunum about 12in. from the duodeno-jejunal flexure, the statement was to the effect that he had to bring the food up right from the very bottom of his stomach, and when one saw the process the sensation could be imagined. The coil of jejunum became more and more distended, and then there were sudden contractions that forced the food out of the coil and back to the duodenum. These contractions were not associated with marked pain, but when the patient said he was going to be sick one saw much more violent contractions that actually cleared the coils almost entirely and swept the contents into the stomach, whence they were voided in the usual manner by the combined effort of voluntary and involuntary muscle.

PYLORIC OBSTRUCTION.

Obstruction is a relative term and depends not only on the narrowing of the canal through which the food has to pass, but on the *vis a tergo*. These are the two main factors, but when the stomach is atonic there is also the static disability resulting from the fact that the lowest part, in which the food collects, is far below the pylorus. In Chap. V, p. 50, I have

* *Proc. Roy. Soc. Med.*, Feb., 1909.

† I have just seen vomiting in association with thickening of the pylorus, probably malignant (No. 2016). In this case, whenever the retching occurred a spasmodic contraction divided the stomach and several extremely powerful peristaltic contractions appeared. These did not move on definitely, and seemed to originate *at the biloculation*. Sometimes they passed in a reverse direction upwards and downwards from the contracted zone at the same moment. Lying down, the appearance of the spasm was almost exactly as in Fig. 47.



Fig. 31.



Fig. 32.

Fig. 31. Marked case of gastropotosis—the tubular form is maintained.

Fig. 32. Radiogram of a typical case of chronic pyloric obstruction, Type 1. The plate was taken 48 hours after the food was given, and the bismuth is seen lying about 5 inches below the level of the umbilicus. None of the food appears to have reached the cæcum. A little more food has been given and can be seen sliding down between the collapsed walls.

discussed the question of the rate of emptying of the stomach and the lines on which this subject can be investigated.

It seems impossible at present, even with detailed notes of many cases before me, to analyse with certainty the various stages, as I have done in the case of œsophageal obstruction—the part that loss of tonic action plays is so difficult to determine. In the large majority the loss of tone is a marked feature, but in a small number perfect tonic action persists in spite of the most striking retention, hardly any of the bismuth food having passed on in 24 hours. Apart from these rare cases, which I shall refer to later, the stages are not so difficult to separate, although there is no definite dividing line between them:—

A. In acute cases.

- (1) It is very seldom that a really acute case is seen. I have only seen two, but in both of these the writhing and twisting of the stomach, in its efforts to pass the food out, was a sight that is not readily forgotten. In both of them it was a carcinoma of the pylorus that was the cause of the trouble. The struggle seems to be over in a short time and the picture becomes that of a chronic obstruction in its third stage, but whether under type 1 or type 2 I cannot tell.

B. In chronic cases.

- Type 1.* (1) Several early cases of obstruction have now been observed, and recognised, in which the compensatory mechanism has been so developed that the emptying of the stomach was more rapid than usual. The waves were of greater power than normal and were noted as of unequal strength. These patients have stated that they were feeling better than usual and our practice in such cases is to re-examine when the patient does not feel so well. In hospital one gives instructions that indiscretions of diet, such as the patient can suggest at once, shall be permitted, and when these patients are again examined one usually finds that there is very definite delay in emptying, and also that the diagnostic character of the peristalsis is quite unmistakable.

Pyloric obstruction

Apart from these features there is another sign that is of very great importance and one that is, I believe, diagnostic of irritation of the pyloric region. This is the rapid secretion of gastric juice which is seen collecting within a few minutes over the column of the opaque food and which can be shaken and splashed (succussion) by moving the patient sharply. The extremely acid nature of this secretion is easily proved by noting the rapid increase of the magen-blase on giving a dose of sodium bicarbonate: Normally the quantity of secretion is quite small, perhaps half an inch in a quarter of an hour, and the evolution of carbon dioxide from it is perhaps a quarter to half an inch, whereas with these cases of hypersecretion the quantity of gas evolved may make the air space increase by as much as an inch in the vertical direction. It is of course impossible to do more than indicate such measurements very roughly; it is, however, a test that can be applied with fair accuracy after a little experience.

- (2) A normal stomach that occasionally shows very powerful waves (or successions of waves) of peristalsis with periods of inactivity between, is suggestive that peristaltic action is becoming worn out, and if this sign is observed on one or two occasions it is practically certain that obstruction is present. I have seen such peristalsis entirely checked both by tr. belladonnæ and sodium bicarbonate, but the action of neither of these drugs is constant in this respect.

There may be some delay in emptying but it is seldom as long as six hours unless it happens to be one of the patient's bad days. These are the cases most frequently met with in private practice and that give most trouble in diagnosis.

- (3) Retention of food is found when the peristalsis fails to empty the stomach, and the patient nearly always complains that he feels his 'food lying on his stomach' for hours. Tonic action is called into play continuously, for the stomach is never empty,

and by degrees the organ becomes atonic. Owing to the failure of tone, the stomach becomes stretched, the muscle is thinned out and cannot produce peristaltic waves of the same power as formerly. Not only so, but the work it is called upon to do in lifting the food to the pylorus increases as the greater curvature sags down towards the pelvis. Even when the stomach becomes quite atonic, its lowest border, sometimes as low as the symphysis pubis, I have very seldom, if ever, failed to see evidence of peristalsis, provided the apparatus was working efficiently. In one of these atonic cases I found quite a quantity of bismuth food in the stomach five days after it had been given, in spite of persistent vomiting. (Figs. 32 and 33.)

Incidentally, it is interesting to note that the heavy bismuth food, lying at the bottom of the stomach, is practically never brought up in these cases—even the most violent vomiting being unable to empty the stomach.

Type 2. I have no evidence whether or not the early stages in these cases are the same as in type 1, but the resulting *x*-ray picture is a most striking contrast. Perfect tonic action is always present and yet the stomach is invariably full of fluid, and presumably this fluid is food. Peristalsis is entirely absent, or there may be an occasional powerful wave. The delayed emptying is just as marked as in the atonic cases, and on examining the operative findings I see that out of eight cases, six showed carcinoma of the pylorus (20, 31, 80, 309, 682, 698), while of the two others one (208) is reported as an extensive ulcer of the lesser curvature close to the pylorus, and the other (313) showed marked cicatricial contraction, the pylorus being one mass of fibrous tissue; precisely similar lesions were found in some of the atonic cases. Fig. 34 is a good example of this type.

Retention of food.

In the latter stages then, pyloric obstruction gives rise to retention of food, and the diagnosis rests on the determination of this fact. The detection of retained food in the stomach when the bismuth examination is commenced is of some importance and will therefore be considered.

If the tone of the stomach is good we can see a broad fluid line below the air, which ripples and splashes when the patient is shaken, even though he has been starved for several hours previously. On giving bismuth food a stream of dense shadows is seen falling from the cardiac orifice down to the lowest part of the stomach—like pitch dropping through water. Naturally, only the lower border is outlined, and on giving more food it is noted that the shadow suggests that the bismuth is only indicating the lower part of the vertical column of the stomach contents. The peristaltic waves are sometimes excessive and when we see very powerful waves that are followed by a period of inactivity, it is practically certain that obstruction is present, but of course all such observations must be confirmed.

When the tone of the stomach is defective the retained food sinks to the lower part and there is nothing, except the way in which the food drops to the lowest part (see Fig. 28), to indicate its presence. The picture is simply that of an atonic stomach in which peristalsis is more violent than one would expect in a thinned-out organ.

Retention of bismuth food is the result of pyloric obstruction and Rieder laid it down that the whole of a bismuth meal should have left the stomach within five hours. As has already been pointed out (pp. 50, *et seq.*) this is too sweeping a generalization, but, with the reservations stated, it is sufficiently accurate. The question of the rate of emptying is, however, discussed on p. 50, but one must again call attention to the fact that in the early stages of pyloric obstruction there may be not only no delay but actually more rapid emptying than usual (as in Case 126)—the result of overaction of compensation.

Bad teeth and septic conditions of the mouth, etc., must

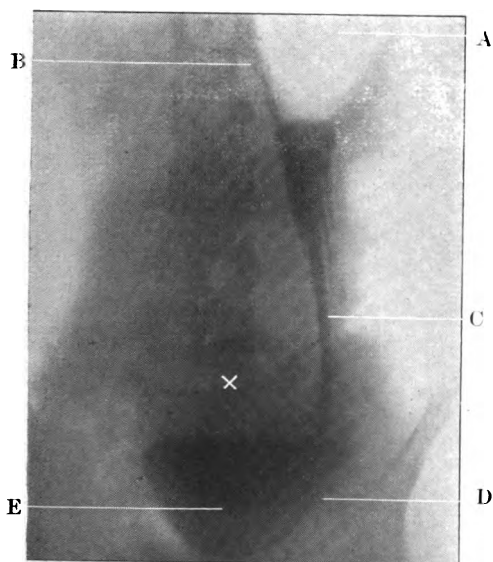


Fig. 33.

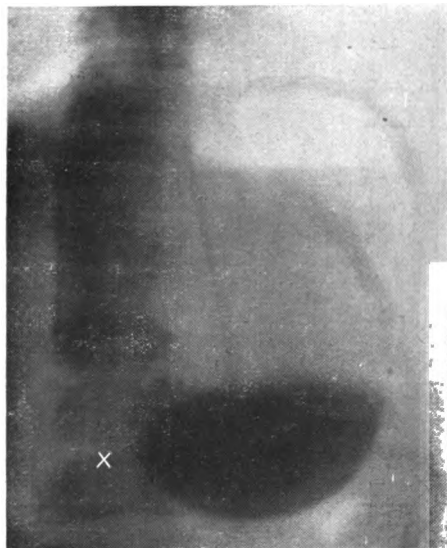


Fig. 34.

Fig. 33. Radiogram of a case of extreme atony in which the lowest part of the stomach extended nearly 6 inches below the umbilicus. A little food has just been given, and is seen extending from the cardiac orifice to the mass of food in the lowest part. Note how this is held up to some extent in the upper part by the walls of the stomach being in apposition. (A) air, (B) cardiac orifice, (C) food sliding down, (D) a peristaltic wave showing as a feeble concavity in the shadow, and (E) the mass of food in the lowest part, (X) umbilicus

Fig. 34. Radiogram of Type 2 of pyloric obstruction. The plate was taken 24 hours after the bismuth food had been given and none of the food seems to have left the stomach. Note the perfect tonic action, the absence of peristalsis, the wide column of fluid above the bismuth shadow and the fluid line forming the lower margin of the air-space.

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be attended to before any reliance can be placed on delay in emptying. I have seen a case cured by removal of carious stumps in which a large quantity of food was still present in the stomach after 24 hours. It is probable that sources of irritation lower down in the alimentary tract may also produce spasmodic pyloric obstruction. I believe that these spasmodic contractions are the direct precursors and probable causes of the formation of actual pathological changes in the stomach walls. (See Chap. XI.)

NOTES ON CASES OF PYLORIC OBSTRUCTION.

(Cases on pp. 154-8.)

The large number of cases that fall under this head is most striking. In 84 out of 270 cases included in this tabulation, pyloric obstruction was verified by the operative findings.

No attempt has been made to separate out the malignant cases from the non-malignant, as so large a number were on the border-line, and nothing but a microscopic examination would have determined the cause of the obstruction. As yet, the *x*-ray examination reveals the mere fact of obstruction, and does not give any definite clue as to the nature of the lesion except when a portion of the gastric cavity is obliterated—such cases have been tabulated in Class v. From the surgical point of view, however, the nature of the disease is of secondary importance as compared with the knowledge that there is obstruction present and that the patient can, almost certainly, be relieved, for a time at any rate, no matter what the cause may be. It is worthy of note in this connection, that in some cases that were undoubtedly malignant, the patient's general condition improved in a manner that was quite as striking as in the non-malignant cases. Not only so but the patients have, in one or two instances, been in perfect health when enquiry was made six months and even two years later.

The large number of instances of spasmodic and organic contractions of the body of the stomach, in connection with pyloric obstruction, is discussed on p. 81.

HYPERSECRETION.—PYLORIC ULCERS.

I have met with a number of cases in which all the appearances of a normal stomach were present but, after perhaps a

quarter of an hour, all the bismuth food occupied the lower part of the stomach, while the upper part contained nothing but thin fluid—the gastric secretion. Such a picture indicates excessive secretion, and is usually associated with irritation, if not actual ulceration, of the pylorus. It is very frequently seen in subjects whose teeth are in a bad state, and I have now seen many such cases in which this feature has gradually cleared up after removal of bad teeth or sinus suppuration. The marked acidity of these excessive secretions is very readily demonstrated by giving sodium bicarbonate and watching the rapid increase of the air space in the fundus. The active secretion, however, cannot be detected when there is atony. Fig. 34 represents the appearance seen when secretion is very excessive, except that peristalsis is present.

My impression is that excessive secretion indicates pyloric irritation and is the forerunner of the formation of a pyloric ulcer. The only point of distinction is that when there is an active ulcer present, one can palpate to a very definite tender spot on deep pressure over the pylorus itself, whereas, in the irritation cases, the tenderness is more diffuse.

The type of stomach also suggests irritation—the peristalsis is rather active and the waves pass more rapidly; sometimes as many as five indentations may be present at once. This type of irritable stomach with active secretion makes one at once suspicious of the condition of the teeth. One has seen many cases cleared up by the dentist; in hospital by removing old rotting stumps, in private practice by taking out bridges that harboured decaying refuse, and covered or were attached to stumps that were actually embedded in abscess cavities. Good-looking false teeth often cover dirty decaying stumps. One woman, in hospital, whom I re-examined eighteen months after her teeth had all been removed, without any shame told me that her plates had only been twice out of her mouth during the nine months she had had them—her symptoms had recurred.

NOTES ON ULCERS OF THE PYLORIC REGION.

(Cases on p. 148.)

The pars pylorica contains comparatively little of the bismuth food at a time and its shadow is therefore not so easy

to see as that of the large mass in the body of the stomach. Moreover, the opacity of the vertebral column is superimposed and renders it almost impossible to make certain of details. As yet, I have seldom seen cases in which I obtained any definite evidence of ulceration from the appearance of the pars pylorica itself, but there are several signs that point towards the presence of an active ulcer in this region. There is no hard and fast line between the cases of ulceration with spasm and those of organic obstruction of the pylorus, and in the following list of cases I have included only those in which there was either no really excessive peristalsis, no delay in emptying, or no marked cicatrization of the pylorus found at the operation.

In some of the earlier cases I detected no indication of any pathological condition. Most of these have remained in their original class (Class iv), *i.e.*, under ulcers of the body of the stomach. It is only in 1910 that I have made the observation that hypersecretion can be easily detected radiographically. The number of cases that have been operated on so far is comparatively small, but in those cases where this phenomenon was well marked the operative findings have shown active ulceration close to the pylorus. Unfortunately there are degrees of hypersecretion, and in the less marked cases the sign certainly does not necessarily indicate ulceration; moreover, the ulceration of malignant disease will give rise to the same sign. On investigating this subject I have found, as expected, that on neutralising the secretion with a solution of sodium bicarbonate, there is apparently a far less profuse liberation of CO_2 in the carcinomatous cases than in those which are due to irritation or ulceration of the pylorus. I have now little hesitation in suggesting a diagnosis of pyloric ulceration if:—(1) the stomach is normal, (2) the peristalsis is active or irregular in strength, (3) no shadows are seen passing through the duodenum, (4) there is very rapid secretion which gives off CO_2 freely, (5) there is a tender spot on deep palpation that coincides exactly with the pylorus.

It will be noted that the picture, as stated, differs widely from that seen in duodenal ulceration (p. 99).

CHAPTER VII.

GASTRIC ULCER AND HOUR-GLASS STOMACH.

(Organic and spasmodic.)

(1) Ulcers of the fundus of the stomach are rare but two cases that I examined came to the post-mortem room. One was a small cicatrized ulcer about two inches from the cardiac orifice on the anterior wall, the other a malignant ulceration of the fundus that did not involve the cardiac orifice. In both of these cases the only symptom noted at the time of examination was œsophageal obstruction and in both there was very marked distension of the lower end of the œsophagus. In one of them a large quantity of bismuth food remained in the œsophagus, and was found there 24 hours later, and this in spite of the fact that the patient had been 'vomiting' and that at the post-mortem there was no trace of obstruction or of any pathological change at the cardiac orifice itself. I think it is highly probable that many of the cases of 'cardio-spasm' recorded by Plummer* and others are in reality due to ulcers near the cardiac orifice.

(2) Ulcers of the pylorus give rise to spasmodic contraction of the pylorus. The actual obstruction in some cases is so marked and so persistent that the stomach may become completely atonic, extending to 4 or 5 inches below the umbilicus, while only a very small quantity of the food is passed out in 24 hours. Later on the ulceration leads to cicatrization but as yet the *x*-ray method does not yield us any information as to the importance of one or other factor in the production of the obstruction. In a few cases of active ulceration close to the pylorus, one has noted very marked and rapid secretion of gastric juice, which, provided the stomach is not atonic, is very easily detected as it lies above the bismuth food. (See p. 69. Note Figs. 26, 34 and 39.)

* H. S. Plummer, *Journ. of Amer. Med. Assoc.*, August, 1908, and June, 1910; J. S. Mayer, *ibid.*, October, 1910.

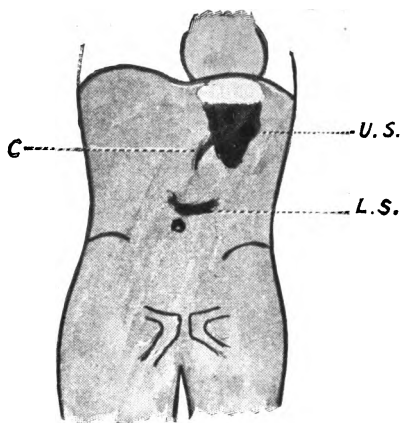


Fig. 35.

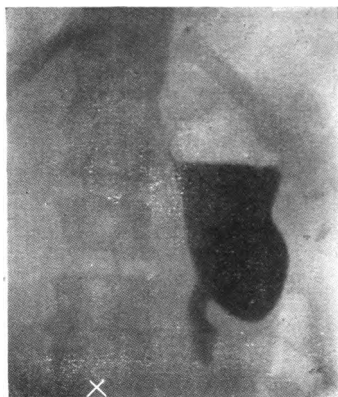


Fig. 36.

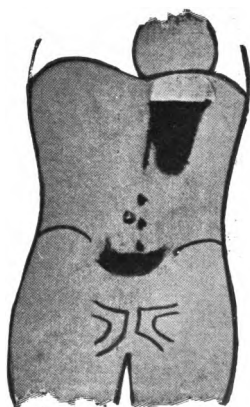


Fig. 37.

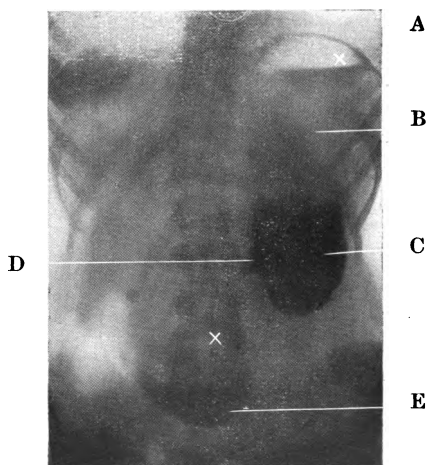


Fig. 39.

Fig. 35. Hour-glass stomach. *U.S.* Upper sac. *L.S.* Lower sac. *C.* Passage leading to lower sac.

Fig. 36. Radiogram of typical upper sac of an hour-glass stomach. This was a cicatricial hour-glass stomach. Plate taken 10 minutes after food was given. None had yet reached the lower sac.

Fig. 37. Hour-glass stomach is often associated with pyloric obstruction. The bismuth food is passing through the channel and falling in drops through the retained food in the lower sac.

Fig. 39. Hour-glass stomach associated with an active ulcer of the pylorus. *A.* Air. *B.* Fluid secreted on top of the opaque meal in 10 minutes. *C.* Lower part of upper sac. *D.* Neck. *E.* Lower sac. *X.* Umbilicus. This case illustrates that the hypersecretion noted with pyloric ulcers is a general secretory activity of the stomach and not localised secretion from the pars pylorica. (Case referred to p. III.)

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Fig. 40.

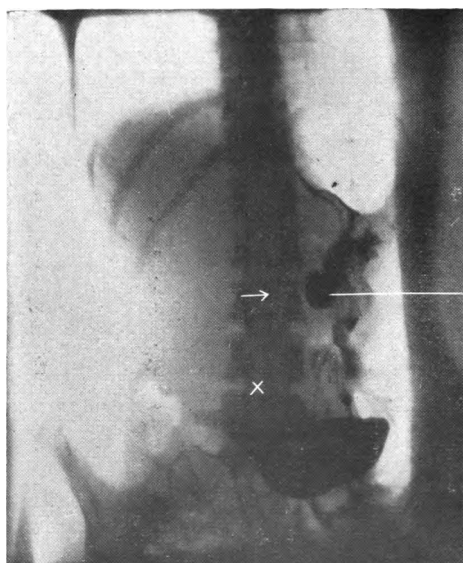


Fig. 41.

Fig. 40. Radiogram of an hour-glass stomach. The opening from the upper sac is not in the usual position on the inner side, but from the posterior wall. Pyloric obstruction is also present, and the food seen in the lower sac had been given 6 hours previously while that in the upper sac had only just been taken. The condition was due to a very dense mass of cicatrization adherent to the pancreas (accounting for the position of the neck of the hour-glass) and also marked thickening of the pylorus. X umbilicus.

Fig. 41. Same case as Fig. 40, 20 minutes later. Palpation showed that the pocket of the upper sac, P, was adherent and immovable.

(3) An ulcer of the body of the stomach is nearly always in an irritable condition and as a result it gives rise to a spasmodic contraction of greater or less severity, the effect, radiographically, being an hour-glass appearance that can only with difficulty be distinguished from the cicatricial hour-glass to which it ultimately gives rise.

The characteristic shape of the ordinary upper sac of an hour-glass stomach is rather puzzling at first, but when one looks at the structure of the stomach one notes that the lesser curvature is far thicker than the greater curvature; consequently any contraction of the circular fibres will result in drawing the greater curvature towards the lesser; the channel between the two sacs is therefore along the lesser curvature, no matter what the position of the ulcer that produces the contraction. One has seen this typical shape resulting from ulcerations in all positions from the lesser to the greater curvature and on both anterior and posterior surfaces. It indicates that no adhesions have formed, unless to the lower border of the liver, or that the contraction has taken place before there has been any tendency to perforation. But in certain cases ulcers tend to perforate and form adhesions with the result that the site of the ulcer becomes a more fixed point, comparatively, than the lesser curvature. The result is that the passage between the two sacs is contracted towards this point. In a few instances one has seen the channel passing out apparently from the greater curvature while in fig. 40 the channel is from the posterior wall, the lesion in this case being an ulcer of the posterior surface that was firmly adherent to the pancreas.

That the stomach is an exceedingly sensitive muscular organ is evidenced by the frequency with which spasmodic conditions are met, quite apart from organic lesions, while it is very rare to meet with any active ulcerative condition that is not complicated by a spasmodic element that, from the functional point of view, is of far greater importance than the actual lesion itself. For instance a small ulcer of the greater curvature may cause an hour-glass contraction that prevents the food passing into the lower part of the

stomach as in cases 207, 389, 400 and 406 where a gastro-jejunostomy had failed to relieve the symptoms.

In three of these cases the evidence of a small ulcer of the body of the stomach had been noted at the original operation, but it had been expected that healing would take place as the result of the gastro-enterostomy. There was thickening of the pylorus and it was supposed that this was the cause of the symptoms, whereas, in reality, it was apparently the insignificant looking ulcer of the body of the stomach that was responsible, if one may judge from the patient's description of his symptoms and also from the fact that all these cases were cured by subsequent operations.

The spasmodic contractions of the stomach most frequently met with are about the junction of the middle and upper third, and the picture given by the bismuth meal is that the food descends to this point and assumes a cone shape. Comparatively suddenly the spasm may relax and the bismuth food passes on into the lower portion, often in a thick stream, giving a somewhat bilocular appearance for a time.

One cannot too strongly insist on the importance of re-examining every case, if possible when the patient is complaining of the symptoms and also when she is free from them, so that a comparison may be made and an opinion formed as to whether the gastric contraction is or is not responsible for the symptoms.

When the bismuth food is held up in the upper portion of the stomach I have very often found that rubbing the abdomen relaxes the spasm so that the contents pass down at once. When the tone of the whole organ is good, the resultant shadow is that of a normal stomach, with possibly a small indentation to mark where the spasm had occurred; but if the tone is defective, as is common in these cases, the contents drop into the lower part, possibly leaving a small quantity at the point where the obstruction was noted. Further abdominal massage usually causes the whole shadow to gravitate to the lowest part, but if traces are observed for a considerable length of time, in spite of massage and a drink of milk, it is practically certain that there is definite ulceration or cicatrization at this point giving rise to the spasm. The

more persistent the spasm in spite of massage, the more probable becomes the diagnosis of actual ulceration or cicatrization. Acute pain on deep pressure at the site of the constriction is very suggestive of the presence of an ulcer.

Case * has noted the presence of a very marked spastic indentation, forming an hour-glass contraction, in cases of duodenal ulcer. I have myself seen this in a few cases only and, on looking up the notes, I find that they have nearly all turned out to be duodenal cases. But the sign can hardly be considered as diagnostic, I think, although Case has noted it sixteen times in duodenal cases.

In a certain proportion of cases small pockets are formed by chronic penetrating ulcers, as pointed out by Haudek,† and these often hold their bismuth food for quite a long time after the rest of the food has passed on (figs. 41, 42). In one patient I examined recently I found a very marked pocket on the lesser curvature (fig. 43). There is no doubt that it was due to a chronic penetrating ulcer although the patient had no gastric symptoms at all. The interesting point in this case is that the patient had no gastric symptoms probably *because* the ulcer did not give rise to any spasmodic contraction, *i.e.*, it was not irritable. In marked contrast to this extremely chronic form of ulceration, I would mention the case of a girl admitted to hospital with subacute symptoms of an indefinite character. Only a few ounces of food could be forced into the stomach, forming a small funnel-shaped shadow, and after 24 hours all this bismuth food was still in the same position. This patient was operated on and there was nothing abnormal to be noted about the stomach except a fairly large ulcer high up on the greater curvature that was threatening to perforate. There was no cicatrization.

These two extreme cases indicate the importance of the spasmodic contraction resulting from ulceration, and all my observations on this subject strongly support Hertz's views of pain due to gastric ulcer: "I believe that tension is the

* J. T. Case, *Journal of the Michigan Medical Society*, November, 1913.

† M. Haudek, "Arch. of the Röntgen Ray," June, 1911.

only cause of true visceral pain." * The importance, therefore, of examining while symptoms are present is obvious.

The penetrating ulcers are nearly always found on the lesser curvature or in close association with the lesser curvature. The symptoms are not as a rule so marked as one would expect with such gross changes. In fact the greatest pain, so far as one can judge, seems to be with the smaller ulcers, away from the lesser curvature, that are associated with great spasm whenever the patient's symptoms are bad. The absence of spasm in some of these ulcers of the lesser curvature is quite definite—in my original tabulation I failed to detect four ulcers only, so far as the operation records go, and these were all on the lesser curvature. Whether this absence of spasm in these cases is responsible for the fact that the tendency to sudden profuse hæmorrhage is most marked in this type of ulcer or not I do not know, but it is certainly suggestive. It is an old saying that ulcers of the greater curvature give rise to hæmorrhage. I believe that the presence or absence of spasm is probably the dominating factor in the arrest of hæmorrhage when once an artery has been eroded by ulceration. If this is so, treatment directed to increase gastric contractions is indicated and, if there is pain, no attempt should be made to relieve it (relax the spasm) till all hæmorrhage has ceased.

Belladonna has a marked influence on some of the spasmodic contractions. Relaxation may be startlingly sudden in some, while in others no effect is noted, although the massage test has proved the condition to be spasmodic. In a very marked hour-glass contraction, which was thought to be partly organic, I found that this drug practically removed the obstruction between the upper and lower parts of the stomach. Acting on the suggestion of this examination, one patient lived in perfect comfort for three years, taking small doses of tr. belladonnæ from time to time. Subsequently however, and without any change in the clinical manifestations, a duodenal ulcer formed and perforated. At the operation the hour-glass

* A. F. Hertz, "The Sensibility of the Alimentary Canal," 1911, p. 47.

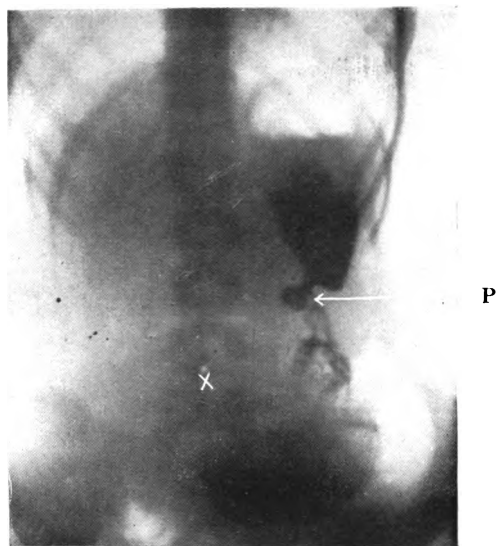


Fig. 42

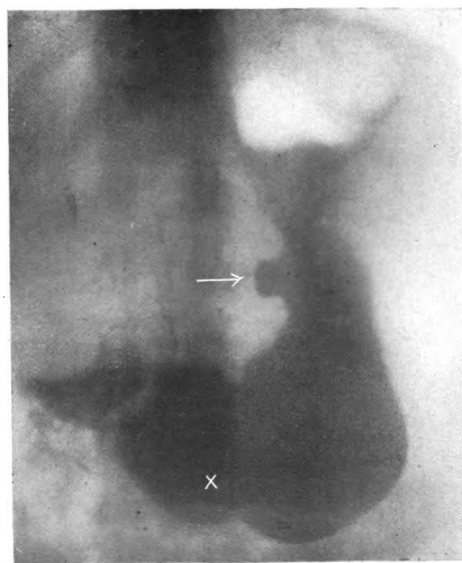


Fig. 43.

Fig. 42. Hour-glass stomach with penetrating ulcer at the neck, P. A plate taken half an hour later showed a little air bubble over the top of the food retained in the pocket.

Fig. 43. Radiogram of case referred to on p. 75. A normal stomach with the exception of the pocket on the lesser curvature, indicated by the arrow, due to a chronic penetrating ulcer (Haudek). X umbilicus

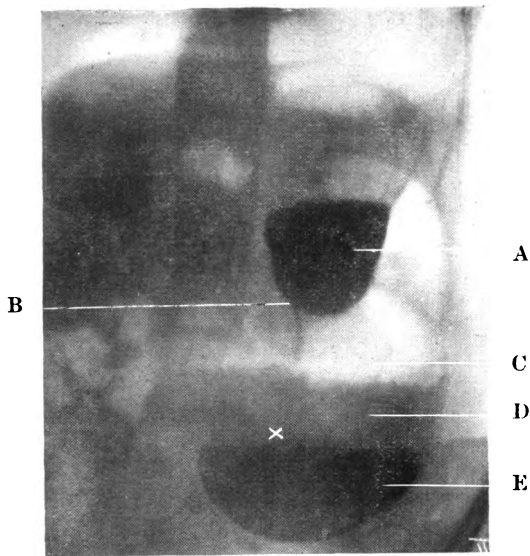


Fig. 44.

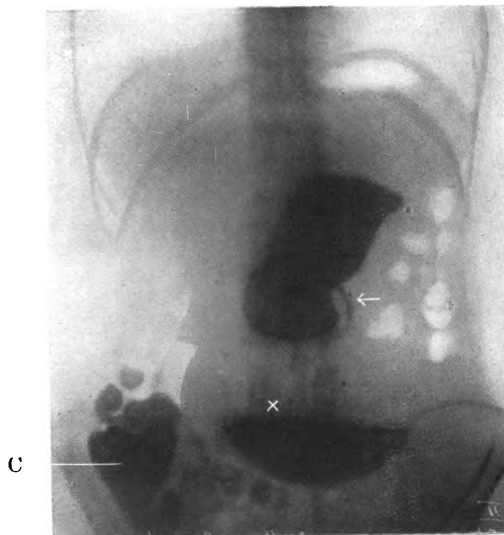


Fig. 45.

Fig. 44. Hour-glass stomach with an extremely narrow neck—no food had passed into the lower sac in 2 hours. The food in the lower sac is that given the previous day, and practically none of it has passed the pylorus. Note the retained ordinary food lying above the opaque food in the lower sac and also the large collection of air above it. A. Upper sac. B. Neck. C. Air in lower sac. D. Retained food in lower sac. E. Opaque food in lower sac.

Fig. 45. Hour-glass stomach with neck opening on greater curvature—due to an ulcer adherent in this region. The stomach was found much distorted. Arrow points to neck. C. Cæcum.

condition was found to be due to a cicatrized ulcer, that was probably active, and was surrounded by adhesions. *Belladonna*, therefore, may relax spasm even in the presence of active ulceration and give rise to a false sense of security, by relieving symptoms that might otherwise be absolutely definite indications for surgical interference.

It is not easy in many cases to distinguish between a functional and an organic hour-glass contraction. I think it probable that the functional contractions are the predisposing if not the actual, causes of the formation of ulcers (see chapter XI). In both cases the bismuth is held up in the upper sac, and it may possibly be noted that the retained food is also present in it. It is always suggestive of a bilocular condition if the lowest part outlined by the bismuth does not reach nearly to the level of the umbilicus without showing some indication of turning to the right, as if towards the pylorus.

On watching carefully it is generally seen that some portion of the shadow is passing on, either in drops or as a thin pencil, and falling into the lower sac. If this is not seen, the patient should be persuaded to take some more of the bismuth meal, or a drink of milk, and this is often sufficient to increase the intragastric pressure, so that the passage will be canalized.

If, on rubbing the patient's abdomen, it is found that the greater part of the shadow descends into the lower sac, the condition is certainly spasmodic. But if manipulation makes little or no difference in the rate at which food leaves the upper sac, a true organic hour-glass condition is probably present. A careful examination of the upper sac will usually reveal the presence of peristalsis on the greater curvature, starting almost under the diaphragm—that is to say, much higher up than usual. I used to consider that the presence of peristalsis in the upper sac was one of the diagnostic points between a true organic and a spasmodic obstruction, but this is not the case: it is seen just as frequently in the spasmodic as in the organic contractions.

It must be emphasized once again that there will be a spasmodic element in almost all organic lesions, and that the

functional disability will not depend so much on the lesion itself as upon the spasmodic contraction to which it gives rise.

Moreover there are occasional cases of spasmodic hour-glass contractions which give all the typical appearances, and yet at the operation there is no gross lesion to be found to account for the spasm. I believe that the spasmodic nature of these cases can be diagnosed if the massage test is thoroughly used. Since I have employed it I have been misled only once, although I have seen several cases that would formerly have been diagnosed as organic lesions. *The greatest safeguard, however, is the re-examination of the patient on one or two other occasions*; spasmodic contractions are not necessarily always present, whereas organic contractions can never completely relax.

Radioscopic palpation is also a very great assistance. It is very seldom, if ever, that one can palpate and move an actual organic hour-glass neck, when symptoms are present, for the patient will resist and tell you at once that you have touched the spot. But in the spasmodic contractions one can usually manipulate the organ without any actual and definite painful spot being located.

In the lower sac the food may show a variety of pictures, but most frequently the shadow is well below the umbilicus, and both atony and pyloric obstruction are present. In 22 out of 38 cases of ulceration of the body of the stomach there was also evidence of a lesion at the pylorus. (See p. 81.)

NOTES ON THE CASES OF ULCERATION OF THE BODY OF THE STOMACH. (*Cases on pp. 149-51.*)

HOURLASS STOMACH.

Ulceration gives an entirely different *x*-ray picture in the body of the stomach to that seen when the pars pylorica is involved, and I have therefore separated these cases from one another.

An hour-glass appearance of the stomach is of quite frequent occurrence, and in the early cases one made many mistakes through not recognising the fact that the stomach is a highly sensitive organ, and is often the seat of spasmodic contractions that may have all the appearance of organic



Fig. 46.

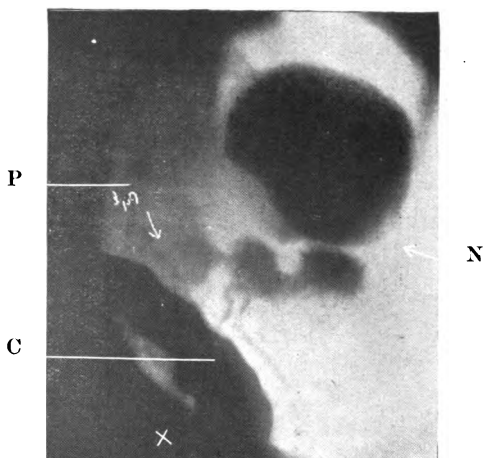


Fig. 47.



Fig. 48.

Fig. 46. Spasmodic hour-glass stomach. It did not relax with massage. No lesion found to account for it, but there was a definite thickening of the duodenum.

Fig. 47. Spasmodic hour-glass that appeared when the patient was lying on the couch. P. Pylorus. N. Hour-glass contractions. C. Colon. Case of duodenal ulcer.

Fig. 48. Upper sac of an hour-glass contraction. Patient had never had any gastric trouble till she swallowed a pin, a week previously. The pin was localised in the stomach at the site of the contraction, but on the following day it was located in the caecum. The hour-glass condition persisted for the 3 weeks she was in hospital. Plate taken half-an-hour after food was given.

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lesions. It was only when I discovered that gentle, or if necessary forcible, massage led to relaxation of spasm, that mistaken diagnosis from this cause ceased to be of frequent occurrence. Tincture of belladonna was also employed, but, although it relieved the spasm in some cases, it absolutely failed in others. Many cases of purely spasmodic contraction have been met with, but there are four to which I wish to draw special attention: Class i, No. 369, and Class viii, Nos. 41, 363, and 398. In these the spasmodic contractions were so marked that I diagnosed the presence of a definite organic lesion, while the operation failed to reveal any underlying cause.

Case 369. A marked contraction near the pylorus that exactly simulated a carcinoma: patient only examined once. The manipulation of the stomach at the operation cured the patient and no trace of the spasm was afterwards seen when he was re-examined, and there was no recurrence of the symptoms.

Cases 41, 363, 398 had been operated upon and the gastro-jejunostomy had failed to relieve the symptoms. In all of them an hour-glass contraction had been noted before the operation, and no trace of biloculation or ulcer had been found to account for it, and when re-examined after the operation the same appearance was noted. All these patients either vomited or retched while under observation and it was evident that it was because of the biloculation, for as soon as all the food had passed down into the lower sac this tendency to vomit ceased and the pain became less severe.

The most searching scrutiny of the outside of the stomach failed to reveal any abnormality, although I think it probable that some minute erosion or irritable point must have been present. In another case (Class vi, No. 713), at which only one examination was possible before the operation, I found a very marked hour-glass in association with an early stage of pyloric obstruction. In this case, although no lesion could be found to account for the spasm, the patient stated that relief of his 'hunger pain' (a typical description) occurred as the food canalized the 'middle

sphincter.' I quite expect to find that this patient is not cured by the gastro-jejunostomy.*

Turning to the records in which a definite ulcer or evidence of old ulceration was found, I have in the following list tabulated 42 cases, and in 38 of them the lesion was indicated radiographically by the hour-glass appearance that was due in part to cicatrization, but to a greater extent as a rule to the induced spasmodic contraction. It is not possible to classify the findings as these have varied widely, from a little cicatrization about an ulcer to a constriction that completely divided the cavity, but the *x*-ray picture has almost always shown a very much narrower channel than was found at the operation. In some cases this has been very marked indeed; quite a small ulcer on the greater curvature with just a little puckering around, has given rise to a contraction so severe and so persistent under massage that only a thin stream could find its way down, and, on the other hand, a well marked cicatricial hour-glass with an indurated ulcer might relax to a great extent with massage, leaving a channel that appeared to be more or less the same size as that found at the operation. There was a greater or lesser degree of spasm in all these cases, and, so far as I can gather from my notes, the severity of the spasmodic element does not depend on the size of the ulcer but rather, one would suppose, on its irritability. Hence the functional disability resulting from an ulcer of the body of the stomach, apart from the stenosis of very marked cicatricial contraction, cannot be gauged by the appearance found at the operation, and, *vice versa*, the extent of an ulceration cannot be gauged from the *x*-ray appearances.

In all these 42 cases there are only four in which an ulcer of the stomach failed to give rise to a contraction that was easily detected on the screen.

No. 24. Large ulcer of lesser curvature near pylorus—one of the early cases in which the pars pylorica was not well seen, and no records of secretion or peristalsis were made.

* See footnote on p. 109.

No. 193. A small ulcer of the lesser curvature, one inch below the cardiac orifice.

No. 418. Large ulcer of lesser curvature. Cicatrices of the duodenum.

No. 760. Cicatrix of lesser curvature, two inches from the pylorus.

It will be noted that in all these cases the ulcer was on the lesser curvature, near one or other of the orifices.

With the exception of these four cases therefore, ulceration of the body of the stomach has given rise to an hour-glass contraction (about 90·5 per cent.).

The association of these hour-glass contractions with pyloric obstruction is very marked. In 14 cases out of the 38 this condition was also present, and it is probable that the four cases in which the gastro-jejunostomy had failed to relieve the symptoms (Nos. 206, 266, 389, and 400) should also be added to the number. Ulceration of the pylorus was also met with in four cases (Nos. 335, 599, 717, 720) and presumably these would eventually have become cases of pyloric obstruction. *In at least a half of the cases of ulceration of the body of the stomach therefore there was also a lesion at the pylorus.*

Among the cases of pyloric obstruction will be found many cases where spasmodic contractions of the middle of the stomach were well marked, but in a far larger number, slight spasmodic contractions were easily recognised as such, and were not recorded. This association cannot be accidental, but whether both conditions depend on some other unrecognised condition within the abdomen, or whether the contraction of the body of the stomach depends on the pyloric lesion I cannot say, but am inclined to the former view.

In two cases (Nos. 418 and 420) cicatrization of the duodenum was also noted.

In four cases (Nos. 207, 389, 400, and 406) a gastro-jejunostomy had failed to relieve the symptoms, and a further operation showed the presence of an organic hour-glass contraction above the stoma, as indicated by the *x-ray* examination.

CHAPTER VIII.

CARCINOMA OF THE STOMACH.

New growths in the abdomen throw no shadows which can be distinguished from the general abdominal opacity; they can however often be demonstrated by reason of displacements of viscera, irregularities caused by the inroads of the growth into the stomach, and by obstruction to the passage of the food, all of which may be shown by giving the patient a bismuth meal. If the pylorus is involved, obstruction may be noted, but this cannot, as a rule, be distinguished from pyloric obstruction due to other causes.

Apart from pyloric obstruction, the diagnosis of carcinoma depends upon the irregularities caused by the inroads of the growth.

The inroads may be quite small, like the outline of a piece of coral, or there may be more or less marked obliteration of the cavity by the growth, which displaces the bismuth, and consequently gives rise to deficiency in the normal shadow. Such inroads may suggest peristaltic waves at first sight, but on further observation it is noticed that they are permanent, and that peristaltic waves sweep up to these notches, are lost to sight, and reappear on the further side of them. In advanced cases the greater part of the gastric cavity may be completely obliterated. (See figs. 49 to 52.) Sometimes pieces of growth displace the bismuth food only partially, so that a thin film of opaque mixture is left. This results in an appearance that suggests thumb marks in the dense shadow.

Growths involving the anterior or posterior walls sometimes invade the stomach so that bismuth is displaced, and a clear space is seen in the midst of the shadow. These clear spaces may appear and disappear as peristaltic waves sweep past, or on pressing the abdomen against the screen, but they must not be confused with similar appearances caused by, say, curds of milk which contain no bismuth. This fallacy, if not detected radioscopically by palpation, is always guarded against by making radiograms on two successive days, and a comparison of these will give



Fig. 49.

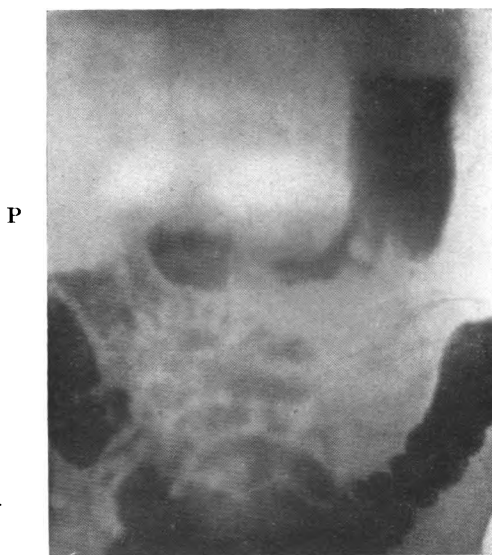


Fig. 50.

Fig. 49. Carcinoma of the stomach. Note the inroads of the growth into the greater curvature. The symptoms suggested growth of the colon only. At the operation the growth was quite inoperable and involved both the stomach and colon.

Fig. 50. Carcinoma involving the lower third of the stomach. No obstruction. P. pylorus. Note the flocculent shadows of food passing through the small intestine.

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the clue to the real interpretation. The obvious fallacy in the diagnosis of carcinoma involving the stomach is, as already suggested, the presence of small or large masses of food in the stomach that are not mixed with the opaque substance. Lumps of bread in the bread and milk may be seen if the food is not properly made, while masses of curdled milk are sometimes noted in pyloric obstruction cases when the patient is on a milk diet. Clear spaces due to such causes are very easily mistaken, and one should always look askance at such an interpretation if one cannot, by radiosopic palpation, feel a corresponding resistance over the suspected area. Indeed, palpation combined with the screen examination is of the first importance in these cases, and will often not only give the interpretation as a neoplasm, but will also yield information that will be a definite guide as to the possibility or otherwise of excising the growth. Of course the information gained is confined to the extent to which the stomach is involved—it seldom gives information as to the secondary deposits in glands, etc.

Adhesions may also cause inroads that are very difficult, if not impossible, to distinguish, but a judicious use of palpation when the colon also is loaded, and inquiry as to the previous history, will nearly always give the clue to the correct interpretation. In this connection one often wishes that it was possible to palpate that part of the organ that is under cover of the ribs.

Spasmodic contractions may simulate the appearance of growths and therefore massage should be employed, and even if there is no doubt a subsequent examination should be undertaken for confirmation. In fact there is hardly a single case in which one is justified in making an absolutely positive diagnosis as the result of one examination. My own practice is to confirm even the most obvious cases by a subsequent observations on the following day. And this is frequently more useful than expected, for the colon contains opaque shadows at this examination and the radiosopic palpation shows the relative movements and displacements in such a way that useful information is obtained.

We cannot exclude the presence of new growths by this

method, but it is seldom that there is no clue to the diagnosis if the new growth actually invades the stomach.

Various tumours of the stomach of a non-malignant character are described, but so far as I know, I have not had the opportunity of examining any.

ABDOMINAL TUMOURS.

Many cases are sent for radioscopic investigation in which an abdominal tumour is felt. Many of these are labelled, clinically, as cancer of the stomach, but it is quite a small number, comparatively, in which the organ is directly involved. If the stomach is found to be free from any evidence of inroads, one at once proceeds to palpation and attempts to establish the relationship of the tumour to the stomach. If this does not yield the necessary information, one gives instructions for more opaque food the same night, so that one will have a heavily shadowed colon to manipulate the following day. One cannot always establish the relationship of tumours in this way, but one is often able to throw some light on the nature of an obscure abdominal case that is of assistance to the physician or surgeon.*

* Apart from the ordinary abdominal tumours the most striking case I have seen was that of a young woman, somewhat stupid in the matter of giving a history, who had an indefinite abdominal tumour that seemed to present in the epigastrium. She was sent for renal examination, and my assistant called me to see the case before using the compression necessary for renal radiography. It was well that he did so, for the screen examination at once showed it to be a case of a large collection of fluid and air in a localized cavity in the abdomen. The stomach, filled with bismuth, was seen to be behind it and the fluid line, with the air above it stretched from one side of the abdomen to the other. On examining lying down, one saw that the air spread out in a large flat bubble all over the front of the stomach, and when one obtained a lateral view one could make certain that the cavity was just below the anterior abdominal wall. The diagnosis was at once clear, as also the patient's history, when one asked leading questions as to the onset, which was with an attack of violent pain after a period of dyspepsia. The condition was due to the perforation of an ulcer and the infection of the abdominal cavity with some gas-forming organism, and the pus that was let off was the most offensive I have ever come across. Clinically the case was quite obscure and the diagnoses had been most varied, partly no doubt because of the patient's stupidity.

P



Fig. 51.

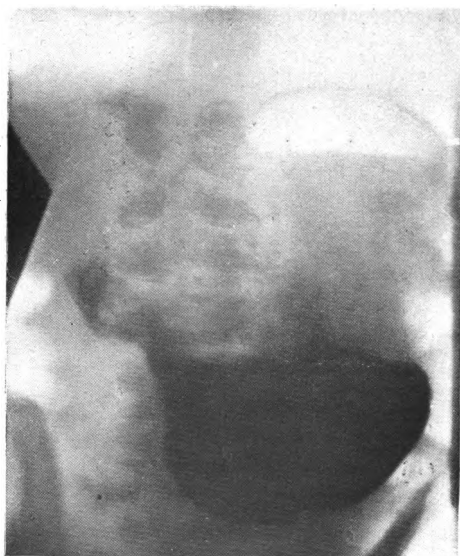


Fig. 52.

Fig. 51. Carcinoma involving middle of stomach and forming an hour-glass appearance. No obstruction. P. pylorus.

Fig. 52. Carcinoma of pyloric end causing obstruction. Note the retained food above the opaque meal. The pyloric portion of the cavity is almost completely obliterated. In some of these cases the shape of the remaining cavity is most irregular.

NOTES ON CASES OF CARCINOMA OF THE BODY OF THE STOMACH. (*Cases on pp. 152-3.*)

To place all the cases of carcinoma of the stomach under one head was quite impossible as such a large number of the pyloric cases were on the border line, and in many cases the surgeon could not tell whether the thickening was due to inflammatory causes or to new growth. All cases of pyloric obstruction, whether due to growth or cicatrization, have therefore been placed together in Class 4, and in the following list are included only those in which the growth invaded the body of the stomach.

In the diagnosis of carcinoma of the stomach reliance must be placed on the displacement of the bismuth food by the growth. In the large majority of cases a considerable portion of the cavity was involved and there was no doubt as to the diagnosis. In a smaller number definite inroads of growth were noted, often difficult to distinguish from peristaltic waves, especially when the apparatus was not working well. Adhesions in some cases gave rise to indentations that were mistaken for carcinoma, and in one case (recorded under Class 1, No. 369) a pure spasmodic contraction gave rise to the same mistake.

An hour-glass appearance is sometimes caused by the obliteration of part of the cavity (fig. 51), and it may be difficult to distinguish a carcinomatous biloculation from the cicatricial condition, as in case 59, but when the growth is sufficiently advanced to give rise to such an appearance, an abdominal tumour can almost invariably be detected.

Clear spaces in the bismuth shadow have been noted on several occasions (case 58 is the only one in the tabulation, but in my notes I have records of eight such observations in patients whose clinical symptoms left no room for doubt as to the diagnosis). The chief source of error is the presence of boluses of ordinary food or even curds of milk, but a confirmatory examination will clear up this point. These clear spaces are often only demonstrated by pressing the patient's abdomen against the screen, and they tend to appear and disappear as peristaltic waves pass over them.

The movements of the diaphragm are sometimes, but not often, restricted, and on many occasions I have found the liver enlarged and the diaphragm pushed up on the right side, indicating secondary growth in the liver. In no single instance have I found secondary deposits in the lungs, in fact the only instance of this complication I have met with in association with abdominal cancer was in a case where I reported the stomach as normal and in which carcinoma of the rectum was subsequently discovered.

Twenty-three cases only are tabulated out of a much larger number in which the diagnosis has been perfectly plain, often both clinically and radioscopically. In many of them the *x*-ray report showed that operative interference would be useless. This probably accounts for the presence of only five cases of this type in the last 300 cases examined.

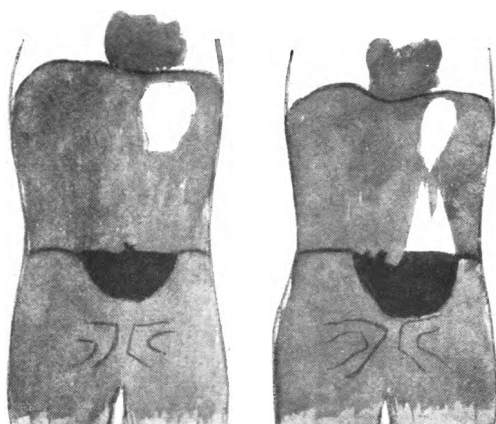


Fig. 53.

Fig. 53. Diagrams indicating the mechanism of gastric borborygmi. In inspiration the air is sucked down, while in expiration it bubbles up over the left kidney between the moist walls of the stomach. (p. 88 and also radiogram Fig. 29.)

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CHAPTER IX.

VARIOUS GASTRIC CONDITIONS.

ÆROPHAGY (*air-swallowing*).

Air-swallowing is not an uncommon condition, and one which may give rise to severe gastric symptoms.

On giving bismuth food the outline of the stomach may be perfectly normal, or it may suggest an atonic condition; but it is always noticed that as the patient swallows his food a certain amount of air passes down the œsophagus with each mouthful. One can actually see the clear spaces of swallowed air, passing down as gaps in the column of opaque food, in the œsophagus. The air space in the stomach increases and may even extend down as far as the umbilicus, unless eructations occur. The cardiac portion may be dilated like a great bubble, occupying the whole of the left hypochondriac region and even displacing the diaphragm upwards. Such patients swallow more air with liquids than they do with solid or semi-solid food, so that any case where this condition is suspected should be tested with a glass of water.

In well-marked cases violent and persistent eructations may take place, and it is noted that the air shadow, instead of becoming smaller, remains the same, or even becomes greater in extent. Sometimes the air distends the lower part of the œsophagus to some extent.

On more than one occasion I have seen the air apparently sucked down the stomach to the level of the umbilicus. It looks as if this is only possible when the lower part of the stomach is anchored down by adhesions, but I have not been able to satisfy myself on this point.

I have noted the occurrence of slight ærophagy in connection with appendicitis on a considerable number of occasions when gastric symptoms were apparently due to this cause.

Occasionally patients are met with who suffer from persistent eructations and yet show no increase or decrease in the

air space of the stomach. When examining these subjects in the semi-lateral position however, one can usually make out that the swallowed air is taken down as far as the cardiac orifice and distends the œsophageal ampulla to a considerable extent. This condition has not, so far as I know, been described and might be termed œsophageal ærophagy.

BORBORYGMI.

The annoying and persistently *recurring* rumbles that some people exhibit are generally supposed to be due to collections of air in the intestine. They are usually located by the patient on the left side about the level of the umbilicus. Sometimes, when the patient can repeat them at will over indefinite periods, ten minutes or more, the bubbling can actually be felt through the abdominal wall, but only when the patient is in the upright position. I have been fortunate in having several patients, all females, under examination by x-rays when they have been suffering from this distressing sign, and on each occasion have been able to locate the exact mechanism of the production of the sounds. They occur *in the stomach* under certain conditions.

As already pointed out (p. 45) the movements of the diaphragm are automatically compensated by a concertina-like contraction of the gastric walls, with the result that there is very little alteration in the appearance, the organ becoming a little wider to allow for the diminished length, but the lower border hardly moving at all, even with forced respiration. But when the stomach is atonic the greater curvature sags down in the abdomen to some inches below the umbilicus and all the food stuffs fall into the lower part, with the result that the walls of the organ come in contact a few inches below the level of the cardiac orifice. This collapsed portion of the stomach separates the air in the fundus from the food stuffs that lie in the lowest part of the organ, and it is kept moist by the secretions. On deep inspiration, in a patient suffering from atony, it will be seen that the compensatory "concertina" mechanism is deficient and the whole organ tends to move with respiration. In extreme cases the lowest part, contain-

ing the food, is resting on the contents of the pelvis and therefore does not move to the same extent. The result is a very marked change in the shape of the air space, the pyriform clear area elongates and extends downwards; in some extreme cases I have seen it extend as far as two inches below the umbilicus, but this is only when there is an excessive quantity of air in the stomach, as in cases of ærophagy. In such subjects the pressing down, or perhaps better the sucking down, of the air on inspiration results in the cutting off of some of the air by the collapsed walls in the middle of the stomach and, on expiration, the air is again free from pressure and bubbles up over the kidney between the collapsed and moist walls giving the characteristic bubbling. (Figs. 53, 29.)

The sounds can be at once stopped by a little pressure on the lower abdomen, *i.e.* by supporting the lower part of the stomach. In fact this was the point that led to the detection of the cause. No sooner was the patient placed in position in front of the apparatus and, as usual, directed to press the abdomen against the screen, than the sounds ceased and all that was observed was that the air space elongated with each inspiration.

The text books that I have referred to, state that the gastric borborygmi are the result of over active peristalsis. This is manifestly not the case for, in these subjects, the peristalsis is comparatively feeble owing to the thinning out of the gastric muscle that is part of the atonic condition with which these sounds are associated.

ADHESIONS. (*Cases on p. 159.*)

For the diagnosis of adhesions we have to depend on the fact that, under normal conditions, it is possible to manipulate the stomach through the abdominal wall, and to determine more or less accurately the fixity or otherwise of the organ. Some cases are more or less obvious, *e.g.*, adhesions of the lesser curvature to the lower border of the liver but, like all other observations on the stomach, one must confirm at a subsequent examination. On more than one occasion I have found that the second examination revealed a

perfectly normal stomach in cases where I had been quite confident that there were adhesions.

In some cases they cause inroads into the gastric cavity that are almost impossible to distinguish from carcinomatous inroads, and in case 699 this mistake was made, while in case 408 the stomach was segmented by a band of adhesions near the pylorus, and other small indentations were noted that suggested this diagnosis, although there was some doubt in my mind as to whether the case was or was not one of carcinoma.

Palpation in the upright position is the most valuable means of determining whether irregularities in outline are due to adhesions or to new growth, for in the latter case one can almost always detect the size and extent of the growth. But with adhesions no definite tumour can be felt, only an indefinite sense of resistance, and, if the transverse colon is full of opaque mixture one can usually obtain fairly accurate knowledge as to the possible relative movements that will be of considerable assistance. A careful enquiry into the history is as necessary in this as in other gastric cases, in order to arrive at a correct interpretation of the radiographic findings.

In case 151 the persistence of atony (or ? gastroptosis), in spite of massage and exercises, led me to suspect the presence of adhesions fixing the transverse colon in the pelvis, as it was impossible to raise the stomach by manipulation although it could be pushed sideways, while the transverse colon appeared to be fixed. The operation in this case showed the presence of an old appendicitis and a cord-like adhesion to the transverse colon.

FOREIGN BODIES IN THE STOMACH.

A great variety of swallowed objects are met with in the stomach, coins, toys, false teeth, nasal tubes, pins, marbles, buttons and so forth, and the only assistance needed in diagnosis is to determine that they are actually in the stomach. This is very readily done by giving a few mouthfuls of opaque food and noting whether it does or does not reach the foreign body. It is very seldom that they give rise to any trouble and in due course they find their way out. On two occasions I have come across cases in which pins gave trouble. In one of them, a healthy girl of 18, the pin, swallowed a week pre-



Fig. 54.



Fig. 55.

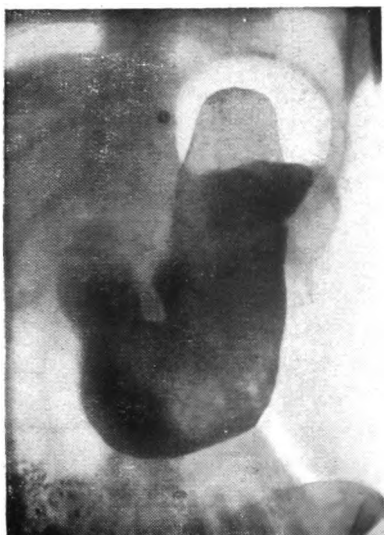


Fig. 56.



Fig. 57.

Fig. 54. Hair-ball in stomach surrounded by opaque food. (p. 91.)

Fig. 55. Ditto, but tumour pressed up from below. Note the displacement of the tumour in the opaque shadow, the top of it coming out above the level of the opaque food.

Fig. 56. Ditto after inflating with CO_2 . The top of the tumour is pressed up into the magenblase and the actual shape of the mass can actually be mapped out.

Fig. 57. The hair-ball after removal

vously, was located in the stomach. On the following day some gastric trouble was present, but the pin could not be found. Opaque food, however, showed a very marked hour-glass contraction of the stomach at the site where the pin had been seen. Again, the day after, the patient was feeling rather better and the hour-glass contraction was not nearly so persistent; it relaxed almost completely with massage. Four days later I again saw the patient and again located the pin, but this time in the region of the cæcum. On re-examining the stomach I again found the hour-glass condition, the gastric symptoms still persisting. The case is of interest and importance from an academic point of view: illustrating the relation of spasm to trauma and of symptoms to spasm.

Hair balls in the stomach are very rare. Figs. 54, 55, 56, 57, represent the only case it has been my lot to meet with. Dr. Ramsbottom and I published the case.* There had been no suspicion of the real diagnosis and this was the first case published in which the diagnosis was definitely made radiographically. The patient, a girl of 28, had an abdominal tumour that was easily palpable and freely movable. It could not be seen on the screen. On giving bismuth food it was at once seen that the opaque food passed to the outer side of the tumour (fig. 54) and down the greater curvature of the stomach so that the tumour was evidently not splenic in origin. In a few minutes the shadow began to canalise down the lesser curvature, which ruled out tumours having their origin on the right side of the abdomen and leaving the certainty that the mass must either be in front, behind, or in the stomach itself. In a few minutes the shadow had extended all round the mass and it was perfectly easy to demonstrate that the tumour was in the stomach itself. Not only so, but one at once saw, by radiosopic palpation, that it was freely movable within the organ (fig. 55) and was not attached at any point to the stomach wall. So far as I know, a hair ball is the only possible diagnosis, and in order to make assurance doubly sure, I inflated the fundus of the stomach with carbon dioxide

* *Archives of the Röntgen Ray*, Sept., 1913.

and could then press the upper part of the hair ball, covered with bismuth food, up into the magenblase. (Fig. 56.) One was able to give an absolutely definite report as to the condition, and even went so far as to give the actual dimensions and a rough drawing of the shape, which were quite accurate. Fig. 57 shows the hair ball after removal, 9 inches long by 3 inches wide, with a tail extending into the pylorus. No history of hair swallowing was obtained from the patient, but her brother said that she had lost her hair completely during an attack of scarlet fever when she was a child.

The pyloric tail of the hair ball made a sharp angle with the body of the tumour that formed, clinically, a distinct notch and suggested the notch of the spleen. The excision was successfully performed by Mr. A. H. Burgess, and the patient made an uninterrupted recovery.

DISPLACEMENTS OF THE STOMACH.

Transposition of viscera is not at all common, and I have had the opportunity of seeing only four cases. Beyond the fact that all the abdominal and thoracic viscera are transposed, there is nothing special to note. In one of these cases, a gentleman suffered from pain in the right iliac fossa. It was known that he had transposition of the thoracic viscera and we were able to demonstrate that the abdominal viscera were also transposed. The appendix was found hanging straight down and quite free in the *left* iliac fossa.

Cases of diaphragmatic hernia with the stomach prolapsed into the thorax have been recorded, but I have not seen any. My partner, Dr. Bythell, however brought an obscure case to our rooms and in this we made the diagnosis of a diaphragmatic hernia, the bismuth food lying apparently above the liver in the pleural cavity. It turned out to be a hernia of the stomach through the œsophageal opening in the diaphragm.

POST-OPERATIVE EXAMINATIONS.

(Cases on pp. 160-1.)

Many patients in whom a gastro-jejunostomy has been performed have been examined, but in this table are included only those in which the operation had failed to relieve the symptoms. In the cases in which a cure was effected I have

not come across a single instance in which the stoma was closed. In a small number, about one-quarter of these cases, the bismuth food was observed passing through both the stoma and the pylorus.

In only one case (524) was the appearance of the formation of a vicious circle noted (*i.e.*, the continued presence of a shadow in the duodenum) and at the operation it was found that the stoma was occluded by adhesions and the duodeno-jejunal flexure kinked. It seems probable, therefore, that the theory of vicious circle vomiting is at least an extremely rare cause of failure, since not a single instance was found in the cases recorded.

In seven cases (44, 416, 470, 471, 483, 524, 530) the *x*-ray examination showed that, either there was obstruction of the small intestine just beyond the stoma or that no food passed through this opening, and in each of these the operation showed the presence of adhesions, the obstruction as a rule being due to kinking.

In seven cases, although the stoma was working perfectly, there was a well marked hour-glass condition present, and in most of them it was evident at the *x*-ray examination that the patient vomited from the upper sac, and that so soon as all the food had passed into the lower sac the discomfort and inclination to vomit passed off. An exploratory operation was performed in six of these cases with the following results:—

41. No case found to account for the hour-glass condition.
Patient unrelieved, and still shows hour-glass condition.
207. Cicatrix of greater curvature with active ulcer, and partial organic hour-glass.
257. Adhesion (extra gastric) forming hour-glass.
266. Cicatricial hour-glass with active ulcer.
363. No cause found to account for the condition. Patient unrelieved.
398. No cause found to account for the condition. Patient relieved five weeks after operation, but this also occurred after the previous operation. On this occasion the appendix also was removed.

In one of the two cases of active ulceration the surgeon noted at the time of the previous operation that a small ulcer was present on the greater curvature, but it appeared to be so insignificant that he did not excise it, in the expectation that healing would take place when the pyloric obstruction was relieved by means of a gastro-jejunostomy. Remarks on the three cases of spasmodic hour-glass condition will be found on reference to p. 79.

In two cases (209 and 357) an hour-glass condition had been found at the time of the operation and the upper sac united to the jejunum. In both of these cases pyloric obstruction also was present with the result that food lodged in the lower sac. Excision of the lower sac was performed in both with complete relief of symptoms.

In case 419 the stoma was situated further from the pylorus than in any other case I have seen. There was marked pyloric obstruction and the peristalsis seemed to squeeze the food into the pars pylorica, and this appearance coincided with the pain. At the operation a growth of the pylorus was found and excised. This relieved the patient, for a time at any rate.

Cases 689 and 756 (entered in Class 6) were both sent for examination because the symptoms persisted, in spite of an operation, at which it was said a gastro-jejunostomy was performed. In both cases the *x*-ray findings were most suggestive of an ordinary pyloric obstruction, and no trace of a stoma could be seen. At the operations it was found that no gastro-jejunostomy had been performed.

In case 10 it was impossible to detect the cause of the delay in emptying of the stomach, which was almost as marked after as before the operation. The stomach was completely atonic and, with the inefficient apparatus then in use, it was impossible to make certain of details in the pelvis.

In case 594 the stoma was working perfectly, and no cause for the persistence of symptoms was seen. On reopening the abdomen it was found that the stomach was adherent to the anterior abdominal wall, and the breaking down of these adhesions brought about a cure.

INCIDENCE OF LESIONS OF THE STOMACH.

It is interesting to note the sex incidence of diseases of the stomach as shown by figures taken from the tables included in this thesis. The extraordinary preponderance of ulcers of the body of the stomach in females is most marked, almost 5 to 1, while from lesions of the pylorus, including both simple and malignant, the male sex appears to suffer most frequently in the proportion of 9 to 7. The male sex also suffers most frequently from carcinoma of the stomach (excluding pyloric cancer), in the proportion of 2 to 1.

The number of cases is comparatively small but the figures are somewhat striking.

	Ulcers of the body of the Stomach.	Pyloric Lesions.	Carcinoma of the Stomach.	Total.
Male	8	54	16	78
Female ...	39	42	8	89
Total	47*	96	24	167

Amongst the cases in which the symptom complex of duodenal irritation was seen (see pp. 99, *et seq.*) there are 30 males and only 8 females. In these cases, although the symptoms were gastric, the lesions were for the most part secondary or referred. If these cases are included in the tables the proportion of incidence of gastric symptoms is nearly equal in the two sexes.

It would appear that success from operative treatment was more probable in men than in women, for out of the 29 cases in which the symptoms had recurred or in which the operation had failed to give complete relief only seven were males while 22 were females, but in two of the latter the subsequent operation revealed the fact that no gastro-enterostomy had been performed.

* Holland (*Liverpool Med.-Chir. Journal*, Jan., 1914) publishes an account of 34 cases of hour-glass stomach. Of these only 2 were in males. In a recent tabulation of my cases I found that out of 71 hour-glass stomachs examined 47 were females and 24 males. Curiously enough there has not been a single male case for 12 months now. In 238 cases of pyloric obstruction 175 were males, 63 females.

CHAPTER X.

SMALL INTESTINE.

After food has been in the stomach for a few minutes, and one has watched the waves of peristalsis pass to the pylorus, there is a small shadow seen collecting just beyond the stomach. It is quite unaffected by the gastric peristalsis, and is known as the caput duodeni. It is more or less triangular in shape and is so very marked that one cannot but think there must be some structural alteration in the walls of the duodenum at this point, about an inch beyond the pylorus. Dr. Moritz, who kindly made sections of this part at my suggestion, was, however, quite unable to detect any anatomical change to account for the appearance. It suggests an auxiliary sphincter, holding a small supply of food to keep an even flow into the second part of the duodenum.

L. G. Cole, of New York, has studied this caput duodeni very carefully by serial radiography and maintains that he can base a diagnosis of duodenal ulcer on variations in its shape and movements. I have not, myself, been able to confirm this work, although one can usually see the caput quite clearly and watch its movements, etc. (see p. 105 a).

When once the food has passed through the first part of the duodenum one loses sight of any definite shadow. The food is mixed with the secretions of digestion and subdivided so rapidly and into such fine division, that one detects nothing but an indefinite mottling until the last coils of the ileum are reached (roughly in 3 hours). For this reason very little is known as to the *normal* movements of the small intestines in man, but, as the result of instantaneous radiography, we believe that they are as indicated by Cannon, *i.e.* (1) irregular peristaltic movements that toss the food forwards and backwards, but in the main forwards; (2) rapid segmentation

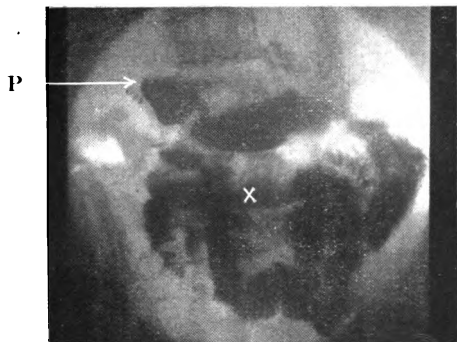


Fig. 58.

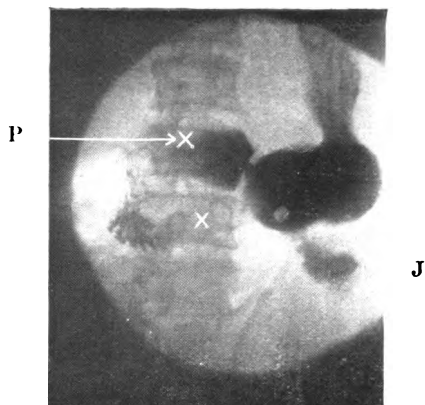


Fig. 59.



Fig. 60.



Fig. 61.

Fig. 58. Duodenal irritation type. Stomach nearly empty after twenty minutes and small intestine overloaded. The coils of small intestine can, however, be separated out easily by palpation. Case of duodenal ulcer and chronic appendix. P. pylorus.

Fig. 59. Obstruction of jejunum. Duodenum outlined and a small mass collected behind the obstruction of the jejunum (J). Shadows were seen tossed back from this point to the duodenum and also possibly into the stomach. Case of adhesions. P. pylorus.

Fig. 60. Small hypertonic stomach commencing to empty very rapidly. Note the shadows in the duodenum and small intestine. See fig. 61.

Fig. 61. Same case as 60, twenty minutes later. Stomach nearly empty and very heavy shadow collected in left iliac fossa. This could not be separated by palpation and collections were still present after five hours. Occasionally, powerful contractions drove some of the food back to the duodenum. Case of early carcinoma of the jejunum referred to on p. 104.

contractions that shred the food and cause eddies into the spaces between the valulæ coniventes.

It is only when there is something abnormal that we see the food clearly after it has passed from the caput duodeni. The class of case in which it is seen passing through the duodenum and first coils of the jejunum is that which I have called duodenal irritation. In these cases there may or may not be actual ulceration present, but the point is that there is abnormal pyloric relaxation, with the result that quite large quantities pass into the small intestine at a time, and we have no difficulty in following the progress for at least some distance into the jejunum. We see the effect of peristalsis in tossing the food backwards and forwards, but there is evidently embarrassment of the segmentation contractions, owing to the overloading by too large quantities being passed on at a time. In some instances this overloading proceeds so rapidly that one suspects that there must be some obstruction causing such heavy accumulations in the small bowel; and yet one can, by careful palpation, separate out all the various coils and find that none of them is distended, but all are well filled. In an hour or less the heavy shadows have found their way to the last coils of the ileum and begin to distend them.

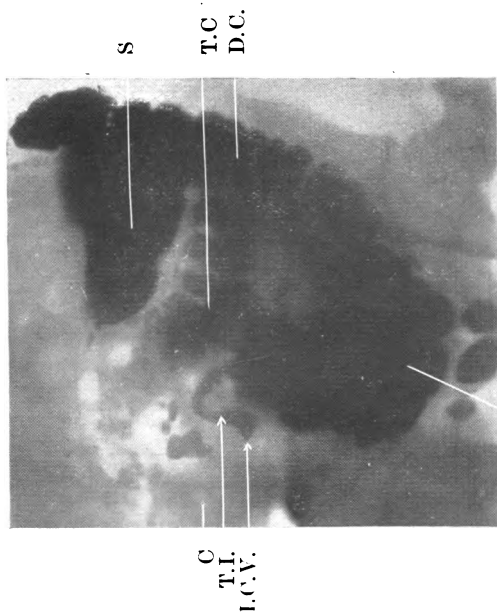
These duodenal cases are most interesting, for they indicate far more clearly than any other the extraordinarily complex nervous mechanism of the alimentary tract with its reflex sensibility. Almost any source of irritation will give rise to this abnormal patency of the pylorus. One sees it in duodenal ulcer—which I believe to be a sequel to this irritability of the duodenum—in the action of ordinary purgatives it is well marked, it has invariably been present in those cases where I have detected lesions of the small intestine, while practically all appendix cases give this sign when gastric symptoms are present. And this pyloric relaxation is associated with lesions even further away, *i.e.*, in the large intestine, and all the cases of mucous colitis I have examined have shown it. Again, one has seen the same appearances in cases of gastric symptoms where there was psychological disturbance and also in the vomiting of early pregnancy. In fact,

one cannot help arriving at the conclusion that there is a nervous mechanism in close relationship with the duodenum, to which disturbances in other parts of the tract are referred. There must be a central exchange, if one may use the simile, in the duodeno-pyloric region that is linked up with other parts of the alimentary tract, and also with the brain, by an intricate mesh-work of nerve fibres of which we know practically nothing. One of the most striking proofs of this is the fact that in certain of these duodenal cases the stomach begins to empty extremely rapidly and the chyme may pass through the small intestine to the ileo-cæcal region in an incredibly short space of time, *i.e.*, in less than half an hour. And yet, if the stomach is not already empty by the time one detects the heavy shadows collecting in this region, one often notes that the peristalsis quietens down and no more food is seen passing out. In fact one may note actual delay in emptying of the stomach, sometimes so marked that one would think it was a case of pyloric obstruction had one not already noted the way in which the first part of the food had passed out. There must be a change in the action of the sphincter to cause this delay; the condition of the pylorus cannot be the same as when the food was first given, for pressure no longer forces food through the pylorus as it did when the rapid emptying was taking place. It is, in fact, a spasmodic pyloric obstruction brought about by a reflex from the ileo-cæcal region—a message to shut off the supply until the surcharge already received has been dealt with. I have termed it the ileo-pyloric reflex.

J. T. Case¹ and A. F. Hertz² have independently shown how the giving, or even the mere smell, of food may bring about emptying of the ileum into the cæcum. It seems as if these two regions, the ileo-cæcal and duodeno-pyloric, are the most important in the sensory mechanism of the tract, and that the rest of the alimentary canal is in close communication. The problems of the alimentary canal are many and difficult to investigate, but the relationship and close linking up of

1. *Am. Quarterly of Rontg.*, Nov., 1912.

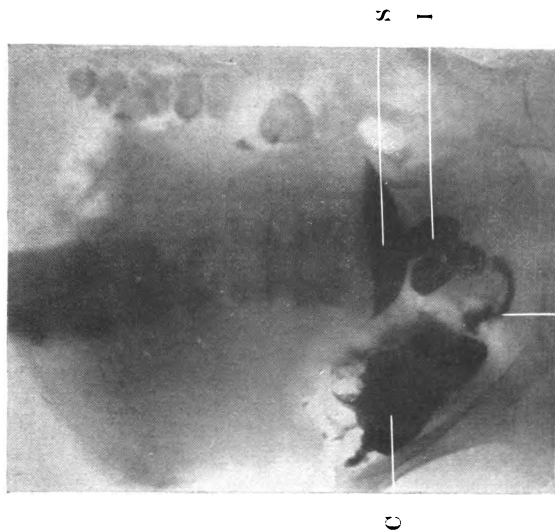
2. *Journal of Phy.*, Oct. 17, 1913.



Ileum

Fig. 62.

Fig. 62. Radiograph of a case exhibiting the ileo-pyloric reflex. At the first examination the typical rapid emptying stomach was seen. The food given at this examination lies in the colon and, as it happens, the caecum is empty. More food was given 6 hours before the plate was taken and half of this is still in the stomach; the rest of it is massed together in the last coils of the ileum. No food could be detected between the stomach and this ileal shadow. None of the second meal has passed into the caecum, *i.e.*, marked ileal stasis.



T.I.
Fig. 63.

Case of appendix adhesions. S. Stomach. C. Position of empty caecum. T.C. Transverse colon. D.C. Descending colon. I.C.V. Ileo-caecal valve. Il. Distended last coils of ileum. T.I. Terminal ileum. (See pp. 54, 98.)

Fig. 63. Delayed emptying. The stomach shows no evidence of peristalsis (S.). Ileum anchored out of position by adhesions. (Coils of ileum (I.). Terminal ileum (T.I.). Caecum of "sloppy" type (C.)

these two centres seems to be fairly well established. In this connection there is another interesting and suggestive phenomenon, *i.e.*, the association of spasmodic and organic hour-glass stomach with pyloric obstruction (see pp. 80, 81); as if the lesions were complementary the one to the other or, more likely, both secondary to some common cause to which we have as yet not found the clue.

DUODENAL ULCER (see also pp. 105 *a* to 105 *d*).

I believe this condition cannot be definitely determined by *x*-ray examination, but there is a symptom-complex which is very strongly suggestive, if not of actual ulceration, at any rate of irritation of the duodenum. The various points that are noted are:—

(1) The stomach always exhibits good tone, even if ptosis is present. Hypertonus is often noted.

(2) The peristalsis is more active than normal, especially when the food has commenced to pass through the duodenum.

(3) The food begins to leave the stomach almost at once and, with moderate quantities, continues to pass out very rapidly until the stomach is empty.

(4) The pyloric relaxation is so complete that large masses of food are seen passing through the duodenum—instead of the fine, almost imperceptible stream that can only be detected with certainty by means of an instantaneous radiogram. In certain cases a separate bolus is seen remaining, apparently in a pocket, in the duodenum.

(5) A painful spot on deep pressure over the duodenum.

NOTES ON DUODENAL IRRITATION.

I have separated these cases out from among those which I had at first classified as normal because, although the stomach appears to be quite normal both radiographically and on the operation table, yet they appeared to show certain radiographic features that I have learned to regard as a

symptom-complex.¹ It is quite possible that my own technique (*i.e.* giving only a comparatively small quantity of bismuth food made with *bread and milk*) is more favourable for demonstrating this symptom-complex than that of some other observers, as one worker (Thurstan Holland, of Liverpool) has told me privately that he sometimes obtains the signs I have described in normal subjects. On the other hand, Hertz² has arrived at the same conclusions and also Rowden,³ of Leeds, who examines Mr. Moynihan's cases.

The symptom-complex consists of the following signs :—

- (1) A normal stomach but always more or less hypertonic.
- (2) Active peristalsis.
- (3) Rapid emptying of the stomach.
- (4) Food seen passing through the duodenum with, or without a persistent shadow in some part of the duodenum.
- (5) A painful spot on deep pressure over the duodenum.

(1) The stomach is invariably 'J' shaped and not a trace of atony is observed although *gastroptosis* is sometimes seen. In fact hypertony is often so marked that it may require quite a large quantity of food to canalize the empty stomach. Such cases are at first suggestive of an hour-glass contraction,⁴ and in two of them (Nos. 97 and 273) I actually suspected the presence of an organic lesion half-way down the stomach, because of the way in which the contraction of the walls resisted the passage of the food.

(The action of gravity is an important factor in canalizing the empty stomach, especially when it is hypertonic, and it frequently happens that at the lowest part of the organ there

1. "B. M. J.," Sept., 1910; "Archiv Rontgen Ray," Oct., 1910.

2. A. F. Hertz. "Sensibility of the Alimentary Canal," 1911, p. 59.

3. B. G. A. Moynihan. *Lancet*, January 6, 1912.

4. J. T. Case has also made this observation and believes this hour-glass contraction to be of frequent occurrence in cases of duodenal ulcer. (See note on p. 75.)

is a considerable pause in the progress of the food before it enters the pars pylorica. H. M. W. Gray, of Aberdeen,¹ interpreted this sign as an indication that there is normally a sphincter at this point, probably indicated by the incisura of His, and on this theory laid it down that the stoma of a gastro-jejunosomy should be made beyond this point.)

(2) *Active peristalsis.* This feature is often not observed till the first food has entered the duodenum. One would not describe the waves of contraction as excessive, but, on the other hand, they are much more active than usual and segment the shadow perhaps three inches from the pylorus. Such peristalsis is seen in early stages of pyloric obstruction, but in duodenal irritation there is no obstruction, in fact the pylorus allows the food to pass on more rapidly than usual.

(3) *The rapid emptying.* The early observations I made on cases of duodenal ulceration proved one point conclusively, namely, that the stomach began to empty itself extremely rapidly and in a manner that was not observed in the normal healthy subject. In some cases a meal of half a pint of bismuth food had left the stomach in less than half an hour, and it was exceptional to find more than a very small quantity in the stomach after three-quarters of an hour. In only one case (No. 66) was any delay in emptying noted, and in this the duodenal canal was stenosed by the cicatrization.²

Not only does the food pass out rapidly from the stomach but it seems to reach the cæcal region in an extraordinarily short time. I noted shadows in the cæcum in half an hour in one case. It is likely that this activity of the whole tract

1. *Lancet*, February 22, 1908, p. 549, and July 25, 1908, p. 224, and December 3, 1910, p. 1610.

2. Although the food begins to pass out rapidly it does not necessarily follow that the stomach is emptied quickly, for in several instances there has been food still present after 5 hours, although no pyloric lesion could be found at the operation; only the duodenal ulcer.—See ileo-pyloric reflex, pp. 98, 93 and 54.

accounts for the frequent clinical history "My food does not do me any good."¹

(4) *Food seen passing through the duodenum.* In the healthy subject I do not expect to see the food passing through the duodenum (except in the caput duodeni)—it passes on in such a fine stream that it is only by means of an instantaneous radiogram that it can be detected with certainty, whereas in these cases one sees quite large shadows pass through the duodenum, and even the untrained observer has no difficulty in tracing the progress from the pylorus to the duodeno-jejunal flexure, although from this point onwards the sub-division of the food proceeds so rapidly that little trace of it is seen on the screen. The appearance is as if there were sudden intermittent relaxations of the pylorus that allowed more food to pass through than the duodenum was capable of dealing with.

It is not after every peristaltic wave that the pylorus opens in this way, nor is it necessarily in response to a specially powerful wave, although it is always as a ring of contraction forces the food against the pylorus that the shadow is seen passing through the duodenum.

In the majority of patients there is a definite shadow of retained food just beyond the pylorus, which appears to be almost continuous with the gastric shadow. When gastric peristalsis is well marked a complete ring is formed as the waves approach the pylorus, cutting off a portion of the shadow which gradually diminishes in size as the constricting ring passes on, the food escaping back into the stomach. The shadow of food retained in the duodenum, on the other hand, is unaffected by the peristalsis, and is always present until the stomach is empty. This small, more

1. Later observations show that it is likely that I should have found delay in emptying in a larger proportion of these cases had I adopted a different procedure, *i.e.*, given a *large* meal five hours before the second examination. One took it for granted that there would be no delay and in these cases instructed for feeding only two hours before the second examination or else re-fed the patient at the time of the second examination.

or less triangular, shadow is known as the "caput duodeni." It is found in the large majority of cases but its position is not necessarily always the same. Hence in some subjects it is best seen with the patient standing, in others when he is lying. Slight rotation of the patient one way or the other will usually bring it into view. In the recumbent position, with a small diaphragm and with the aid of the wooden spoon, this duodenal cap can be carefully watched and, if there is any deformity, the palpator can be so used as to detect whether this can be altered by pressure or manipulation. Pockets may be formed as the result of cicatrization and, in these, traces of opaque food may be found for some time after the stomach is empty. It is stated that post-pyloric ulcers account for over 90 per cent. of duodenal ulcers; hence it is important to detect the presence of cicatrization in this region.

But it must not be assumed that because there is evidence of deformity of the duodenal cap that this is necessarily indicative of an active ulcer—granted that we arrive at the conclusion that these deformities of the shadow of the food within the duodenum are permanent, it does not tell us that this is due to an active ulcer. Cicatrization is the result of ulceration, and its presence does not necessarily indicate that the trouble is still active. Deformity of the caput duodeni, and the presence of opaque food in pockets in the duodenum, are more likely to be found in patients who have suffered from duodenal ulcer, than in those who have recently become the victims of this trouble. Those who suffer from this complaint give a history of recurring attacks, each attack, presumably, coinciding with a recurrence of the ulceration—the effects of cicatrization will be just as evident when the patient is in perfect health as when an active ulcer is present.

(5) *Pain on pressure* over the duodenum is suggestive, but if a point of maximum pain is found on deep pressure on to the exact site of the suspected deformity of the shadow, or pocket of retained opaque food, then I believe one can be fairly certain of the presence of an active ulcer. (See pp. 105 *a et seq.*) The presence of very definite and well defined duodenal irritation is also strongly suggestive of the presence of active

ulceration or, at any rate, of a condition in which ulceration is likely to occur.

Exceptions and anomalies.

In two cases only (Nos. 240 and 241) a duodenal ulcer was said to be present (by the same operator) when the symptom-complex was not noted, but case No. 241 died from hæmorrhage, apparently from the duodenum, a few days subsequently, and at the post-mortem not only was no cause found for the bleeding, but also there was a complete absence of even a suspicion of ulceration of the duodenum, although grey patches were observed on the peritoneum. Case 241 is therefore placed under the normals, while case 240 remains in this class, although somewhat discounted by the post-mortem on case No. 241. Slight atony was noted in only one case (No. 501) although all the other features were well marked, while gastropsis with absence of all the features of the symptom-complex occurred in case 730.

In all these cases a certain train of appearances was established, and on analysis I found :—

Duodenal ulceration	-	-	-	14 cases.
Cicatrization of the duodenum	-			7 „
Adhesions about duodenum (generally in connection with gall-bladder)				13 „
Carcinoma in this region	-	-	-	3 „
Appendicitis, abscess	-	-	-	1 „
Appendix fixed by adhesions near to duodenum	-	-	-	1 „

I used to record only the actual gastric lesion in my own notes and did not appreciate the possible importance of evidence of old appendicitis or other abdominal inflammation as a possible primary cause of the trouble.

It seems probable that many different lesions in the abdomen may give rise to what I believe to be irritation of the duodenum, and will give this symptom-complex although it is most frequently found in association with actual pathological lesions affecting the duodenum itself.

It often looks as if the 'duodenal irritation' was the result of some other lesion within the abdomen (as in case 658), and that the presence or absence of actual ulceration was more or less accidental. This is entirely in accord with Moynihan's view:—'I have long held the view that the diseases of the stomach, duodenum and gall-bladder, with which the surgeon deals, are not primary but secondary.' (*Lancet*, Jan. 6, 1912.) It is for this reason that I have termed this the symptom-complex of 'duodenal irritation,' as I feel confident that further investigation will prove that duodenal irritation and ulceration are generally, if not always, secondary conditions.

In case 658 the symptom-complex was found in association with a tubercular ulcer of the jejunum, this lesion being discovered at the operation in consequence of the *x*-ray report, while in a more recent case the same appearances were seen in connection with an early carcinoma of the jejunum about twelve inches from the duodeno-jejunal flexure. This case is of particular interest as the symptoms were extremely vague, and it was purely on the *x*-ray diagnosis of a lesion of the jejunum that the operation was undertaken. Unfortunately the patient died, from delayed chloroform poisoning it is supposed, and the condition of the mucous membrane of the duodenum showed that there had been some general inflammation (duodenal irritation) of which no trace could be seen on the peritoneal surface.

(January 1913.) Since writing the above, 4 more cases of lesions of the small intestine have been operated on, and in each of them I had noted a very marked duodenal symptom-complex as well as the accumulation of shadows in the small intestine resulting from the obstruction and defective movement caused by the lesion. I have also noted the symptom-complex several times in association with mucous colitis. In a recent case it was very markedly seen in a patient who had a mouthful of septic teeth, and it was when looking for a cause for the *x*-ray appearances, which gave one the impression of secondary gastric trouble, that the teeth were noted. At the operation there was absolutely no evidence of present or past disease to be found in the abdomen. It is also present, to some extent at any rate, in appendicitis, if the symptoms

are at all gastric. I also noted similar appearances in a lady who had quite severe symptoms, and in this case again one had the impression that the symptoms were secondary. A few weeks later the patient (aged 45) developed slight delusions and her gastric symptoms disappeared.

The multiplicity of conditions then, in which duodenal irritation, or even ulceration is noted, makes one more and more convinced that, in the large majority of cases, the duodenal trouble is secondary. It is as if the duodenal region is the storm centre for the alimentary tract to which disturbances in other parts, or possibly even in the nerve centres, are referred, in many cases before any local manifestations have developed.

OTHER LESIONS OF THE SMALL INTESTINE.

In tubercular peritonitis and other forms of adhesive peritonitis, I find that the segmentation contractions are defective, with the result that definite shadows are seen in various coils of small intestine. Several instances of lesions of the jejunum have been met with and in each of them, besides the evidence of obstruction, a well-marked duodenal symptom-complex has been noted.

Definite lesions of the small intestine causing symptoms are not common, but in previous paragraphs I have already referred to some of the cases met with (p. 104). The x-ray appearances are those of an obstruction, and one cannot go further than this as a diagnosis without careful consideration of all the other sources of evidence. In every case one's observations must be confirmed and full use made of radio-scopic palpation, both in the erect and horizontal positions.

THE "POSITIVE DIAGNOSIS" OF DUODENAL ULCER.

Lewis Gregory Cole* has written on several occasions on the diagnosis of post-pyloric ulcers by means of serial radiography. There are now several writers on the subject including Ariel George and Isaac Gerber,† who, following on the

* Arch. Röntgen Ray, April 1912, Oct. 1912. *New York Med. Journal*, May 1913, *Lancet*, May 2, 1914, etc.

† Am. Quart. of Röntgen, Dec. 1913.

same lines, speak of the "positive diagnosis of duodenal ulcer," and complain of the "lack of positiveness" in the writings on this subject.

Apart from the complicated apparatus and excessive cost, I have a great admiration for the thoroughness and accuracy of the work that these and others have given us. Their work is certainly positive and tangible. Nevertheless, although they express their opinions with such convincing precision, yet there is a great deal to be said for those of us who are more or less content with our "symptom-complexes" and "lack of positiveness." When surgeons, who actually see and feel the duodenum, cannot be certain whether or not there is ulceration present unless the peritoneal surface is involved, when one has actually seen both the positive and negative diagnosis of the surgeon confuted in the post mortem room,* I, for one, can never feel justified in making sweeping and dogmatic assertions on this subject.

"The duodenal ulcers that consist of merely a mucous membrane erosion, of course, will not show any effect upon the bismuth, but these are an almost negligible portion of the cases and they probably give only slight and transient symptoms. These are the cases, which are of no surgical consequence.†"

Such a paragraph indicates that the writers do not grasp the fact that the danger from a duodenal ulcer does not lie in the deformity it produces but in its erosive qualities. The bleeding from a small superficial ulcer may be just as serious as from a large one, while the chances of perforation are infinitely greater when cicatrization has not occurred than after this natural barrier has been formed. One cannot say that ulcers of this type are "of no surgical consequence" (? because we cannot make a positive diagnosis radiographically) when fatal hæmorrhages have occurred from such lesions, even though the symptoms, apart from hæmorrhage or perforation, may be slight and transitory. Moreover, it must not be forgotten that every ulcer has its beginnings in small things, maybe only an erosion of the mucosa. It is only when the lesion has become comparatively chronic that thickenings occur, whether on the

* See note on p. 103, Case 241.

† Am. Journal of Röntgen, May, 1914.

mucous or peritoneal surfaces—it is in the early stages, often before definite symptoms have developed, that the ulcer is an acute danger to life; in the later stages it gives rise to the constantly recurring disturbances of digestion, sometimes accompanied by hæmorrhages, that we all know so well, thanks to Moynihan and other surgeons.

Turning now to the radiographic side of the picture.

In the first place, to my mind, the presence or absence of an actual duodenal ulcer is more or less accidental whenever duodenal irritation is present. An ulcer may or may not be formed; it is a sequel in a predisposing condition of the mucous membrane. But the more marked the duodenal irritation the more likely is an ulcer to occur. I believe, and have strong evidence to support the view, that the symptoms ascribed to duodenal ulcer (and usually cured by operation) are the result of duodenal irritation and not of the ulcer itself. If this is so, we may have a “clinical duodenal ulcer” without any definite and constant deformity of the shadows in the duodenum on which this positive or negative diagnosis is based.

Secondly, the well-known clinical fact of the tendency of duodenal ulcers towards healing and recurrence postulates the formation of an ever-increasing scar. It is only reasonable to allow that the scar may produce just those deformities and pits on which this so-called positive diagnosis of ulceration depends. It is rather the effects of ulceration that will be demonstrated by this method, and there is not, I hold, any clue given as to whether this deformity is or is not of *present pathological significance*. And this is the crux of the question, for although the surgeon may be interested in evidence of old duodenal trouble, yet it is no guide for operative interference. He does not require to operate on cases when nature has already completed the work, but he certainly does want to know whether or not it is likely that the conditions that led to the formation of the ulcer are still present. If we can answer this negatively, then the case is one for the physician, but if our answer is in the affirmative, it ought to be a definite guide to him in deciding what course to adopt.

My third point hardly affects the argument, but is, rather, the explanation of my own neglect of this particularly interesting and well worked out procedure in diagnosis. As already stated, I believe the formation of an ulcer is a more or less accidental sequel to a state of duodenal irritation. And, I am convinced, this state of duodenal irritation is itself a secondary manifestation. That a gastro-jejunostomy happens to relieve the symptoms in a large number of these cases is no argument either for or against this view, and, as we come to recognise the causes of duodenal irritation, and eliminate them one by one in each case, there will be less and less need of operation. My point therefore is that it is not the detection of the ulcer that matters, but, given a condition that may lead to ulcer, *i.e.*, duodenal irritation, we should direct our attention to seeking and removing the cause or causes of this condition. I believe that eventually the operation of gastro-jejunostomy for this trouble will be a confession of weakness, for it will indicate that we have failed to find and remove the cause of the recurring attacks.

It is not in any spirit of carping criticism that this note is written—nobody would be more pleased than I if it were possible to be as certain over the diagnosis of duodenal ulceration as we can be about such definite and concrete problems as urinary calculi.

CHAPTER XI.

THE ÆTIOLOGY OF GASTRIC AND DUODENAL ULCERS.

GASTRIC ULCER.

The ætiology of gastric ulcer has always pertained to the department of the pathologist. Unfortunately the pathologist can only study the dead subject and, as we know, the stomach as it fulfils its functions differs widely from the flaccid sac that we see in the post-mortem room or even on the operating table. That morbid anatomy does not always give a true picture of the cause of symptoms has been indicated on many occasions and in the pages of this book there are references to several cases that bear upon the point. For instance on p. 32 a case of obstruction of the œsophagus is described in which the patient was almost dead from starvation and the œsophagus was greatly dilated. Yet the post-mortem showed no sign of the obstruction that had been almost complete nor of the dilatation that had followed, and this is an extreme case in which the bismuth food was still found in the œsophagus after 24 hours. Instances of spasmodic contractions of the stomach of which no trace could be found at the operation are very numerous. Special note is made of three cases on p. 79 in which a gastro-enterostomy had failed to relieve symptoms and in which a very marked and persistent hour-glass contraction was noted. In all these cases the patient vomited from the upper sac and it was evident that the hour-glass contraction was the cause of the trouble, and yet no pathological change could be found to account for abnormalities that were causing symptoms which made life a burden. It is clear that in many cases morbid anatomy tells but half the tale, that half which relates to tissue changes and infers that the functional disturbance is due entirely to these changes. As I have already said (p. 73) it is often the smaller ulcers (just as it is often the smaller renal calculi) that give rise to the severest symptoms. On the other hand, cases have been met with in which gross pathological changes have certainly been

present in the stomach wall with practically no symptoms. A chronic penetrating ulcer is mentioned on p. 75 as giving, and of having given, practically no trouble. One of the most completely fibrous hour-glass contractions I have seen, had not given rise to very severe symptoms. In fact the morbid anatomy does not necessarily give the clue to the actual disturbance and, apart from the patient's symptoms, we have no guide to the functional effects of a lesion of the stomach or intestines except by the *x*-ray method of examination which in its turn has its own limitations; although it shows us the functional results it is only by inference that we diagnose the underlying cause, attempting to separate the factors of spasm and morbid process that have produced the effect.

It was while reading the final proofs of the first edition of this book that the ideas embodied in this chapter forced themselves upon me. The interpretation of the pathology of the living assumed an importance that I had not previously attached to it, even though I was so fully conscious of the importance of the spasmodic element as a complication of organic lesions. And this is just the crux of the whole argument. At the operation one saw a small ulcer of the stomach, whereas at the *x*-ray examination one had seen a typical and persistent hour-glass contraction which must necessarily have been spasmodic. One took it for granted that it was cause and effect, the ulcer being the cause and the spasmodic contraction the effect produced. And yet, on looking back, one found cases where we had the effect apparently without the cause, the spasmodic contraction without ulceration, and it was this that set me thinking that perhaps cause and effect had been confused and that possibly the ulceration was the effect of the spasm, and that the spasm was due in the first place to some other cause, although it could also be produced by ulceration.

It was not a case of evolving a theory and setting to work to prove it; the hypothesis forced itself upon me and it was simply a case of analysing the various impressions that have, I believe, opened up the solution of a problem that I had no thought of investigating. It is hardly likely that my observations cover the whole of the ground, but I am fully convinced

of the accuracy of the deductions as far as I have been able to carry them during the few weeks that have elapsed since I arrived at this new conception of their significance.

The observations on which the deductions are based are as follows :—

(1) The stomach is very sensitive and many cases of spasmodic contraction have been met with ranging from those that were readily relaxed by massage to contractions that were actually mistaken for organic lesions. Quite a large number of spasmodic hour-glass stomachs have been examined and found at operation to show no trace of ulceration or other abnormality that, by local action, would cause the spasm. These spasmodic contractions have been met with in the body of the stomach and also at the pylorus. On several occasions I have seen that the vomiting took place from the upper sac and as soon as the food passed down the tendency to vomit, and the pain, ceased. They were often present one day, absent the next; they could usually be relaxed by massage or manipulation to some extent and were not necessarily found at subsequent examinations. There was no pain over the site of the spasm on deep palpation.

(2) Identical contractions have been met with, in fact are almost invariably met with, in association with even quite small ulcers, and I have always believed that the severity of the spasm resulting from an ulcer was dependent on its irritability and not on the size of the ulcer, since small lesions gave just as, if not more, marked contractions than the large florid ulcerations.

(3) I have seen pyloric obstruction—as indicated by seeing the greater part of the food still in the stomach after 24 hours—permanently *cured* by removing bad teeth (p. 68).

In an out-patient with many decayed teeth, practically the whole of the food was still present in the stomach after 24 hours. On admission he was made to use a tooth brush and, when I examined him a few days later, practically all the food had passed out of the stomach in five hours. He was a fairly intelligent man and had tried careful dieting before he was admitted to the hospital with no effect.

(4) In some cases I have been so much impressed by the

irritable appearance of the stomach that, in spite of fairly well-marked clinical evidence, I reported that I thought the symptoms were secondary, the condition of the teeth being, in my opinion, sufficient to cause the radiographic appearances noted; this in spite of the fact that retention of food had been noted. In none of these cases was any evidence found at the operation of gastric trouble: in one or two, however, there was evidence of old appendix inflammation.

(5) An irritable condition of the stomach, or even an hour-glass contraction, was frequently noted when there was severe constipation.

(6) Spasmodic contractions, forming hour-glass stomachs have been noted as being not so marked or entirely absent after the bowels have been moved in cases of severe constipation.¹

(7) In taking brief clinical histories of approximately 1400 cases I have been struck with the frequency of a history of constipation, and also by the presence of bad teeth or a history of having had bad teeth removed after the onset of the symptoms.

(8) In fully a half of the cases of ulceration of the body of the stomach there was also evidence of retention of food behind the pylorus and in the large majority of these latter there was actual thickening of the pylorus. In two cases in which no thickening of the pylorus was detected at the operation, a gastro-jejunostomy to the upper sac failed to cure and a subsequent examination showed the bismuth retained in the lower sac. A further operation showed well-marked thicken-

1. Case 713 (quoted on p. 79) is instructive in this respect. There was a very marked hour-glass condition in association with pyloric obstruction. Nothing was found to account for the hour-glass contraction, but the pylorus was thickened and therefore a gastro-enterostomy was performed. I expected that this patient would not be cured, but a year later he writes: "I have put on two stone in weight and can eat everything. I was always very costive, in fact I never had a motion without opening medicine, but I have never had a dose since the operation." In the light of other cases it looks as if the operation, by allowing more food to pass, had relieved the constipation, and that this was the cause of the spasmodic hour-glass contraction.

ing of the pylorus, and excision of the lower sac cured the patient.

(9) In a very large number of the cases of pyloric obstruction a more or less marked spasmodic contraction was noted in the body of the stomach. The relationship between spasm or even ulceration of the body of the stomach and pyloric obstruction is so marked that it can hardly be accidental. I have often remarked that it looked far more as if lesions in these two sites were dependent on some common cause than on one another.

(10) Moynihan put into words a belief that had gradually been taking shape in my own mind—"the diseases of the stomach, duodenum, and gall-bladder, with which the surgeon deals are not primary but secondary" (*Lancet*, Jan. 6, 1912). I had been much impressed by what I called the interdependence of abdominal conditions, as when one found an irritative condition of the large bowel, *e.g.* mucous colitis, one usually found the appearances, and often the symptoms, of duodenal ulcer, while in one case there was a gastric ulcer with hour-glass contraction. The duodenal appearances and symptoms were also noted in several cases of lesions of the small intestine (carcinoma, tubercular ulcer and adhesions) in which the actual site of the disease was indicated to the surgeon solely by the *x-ray* examination. In one of these, a typical ulcer of the duodenum was also found, while in another, who died a few days after the operation, the mucous membrane was injected and inflamed, although there was no evidence of this on the peritoneal surface. In appendicitis, when there were gastric symptoms, these same appearances, indicating duodenal irritation, were also noted from time to time at the *x-ray* examination.

(11) The excessive and rapid formation of gastric secretion has been noted in all the later cases in which a pyloric ulcer has been found, except those in which marked retention of food was also present, this feature making it impossible to detect the hypersecretion. Well-marked hypersecretion has been noted in cases where there was slight delay in emptying (six hours), and under medical treatment not only has the delay in emptying disappeared, but also no hypersecretion

could afterwards be detected. The hypersecretion seems to be the accompaniment of pyloric lesions and disappears with the pyloric "irritation." I have not yet seen hypersecretion in association with an ulcer of the body of the stomach unless there was an actual lesion of the pylorus also present.¹ In one instance I found what was evidently the upper sac of an hour-glass contraction of cicatricial type, and during the half-hour I had the patient under observation (the surgeon was waiting to operate) only a very small quantity of food found its way through into the lower sac, but there was profuse secretion into the upper sac and, as suspected, this indicated an active ulcer of the pylorus. (Fig. 39.) At the operation there was only a very narrow channel connecting the upper and lower sacs, so that the secretion must have been poured out from the cardiac end, suggesting very strongly that the hypersecretion of pyloric ulcer is a general secretory activity, probably reflex, and not a local hypersecretion dependent on the direct irritation of the ulceration. Whether the pyloric lesion is the result of the hypersecretion or *vice versa* one cannot say, but I strongly suspect that they are both dependent on a common cause and not on one another.

(12) In operations for gastric and duodenal ulcer there is very frequently evidence of old appendix trouble.

(13) Gastric symptoms—the old symptom-complex of gastric ulcer—have been cured by short circuiting the large intestine in a case of severe constipation. (*Lancet*, February 8, 1893. Paris correspondent.)

(14) My personal experience of oral sepsis is instructive. Occasionally, when I am *below par*, I have acid risings into my mouth, probably indicating an excessive formation of gastric juice, such as I have frequently noted in association with pyloric ulcer in marked cases, with "pyloric irritation" in the less marked cases.

On two occasions I have been conscious of retention of food for a prolonged period, and once I actually brought up a mouthful that contained fragments taken on the previous day. A more thorough and persistent use of an antiseptic mouth

1. If the stomach is emptying rapidly one has not the same chance of detecting the presence of hypersecretion.

wash relieved this unpleasant symptom within the course of a couple of days.

Occasionally I am also troubled with slight hunger pain.

I am certain I do not suffer from any gastric lesion for I have perfect digestion in the ordinary course of events, and do not know what indigestion is! nor does the radiographic examination reveal any abnormality.¹

(15) The experimental production of gastric ulcer in animals, shows that these always tend to heal very rapidly and a typical chronic ulcer has not yet, I believe, been produced, even by causing local thrombosis.

(16) I know that there is considerable literature on the subject, but I have seen no case in which ulceration has taken place at the site of a gastro-enterostomy; and yet at this point the mucous membrane is comparatively roughly joined, and one would certainly expect that, if ulceration was caused by the action of the gastric juice, this would be a *very common* sequence to the operation, especially when we remember that in most of the operations a clamp is used that must produce a certain amount of bruising. This is certainly not the case in the hospital with which I am connected,² and it is inconceivable that the mucous membrane is brought into perfect apposition in every case.

Taking all these observations together, I think that they become intelligible on the following hypothesis:—

Septic conditions in the mouth or other sources of swallowed septic matter, constipation, mucous colitis and a variety of other conditions are capable of producing spasmodic

1. It is now a year since I had all suspicious teeth removed, and I have never had any recurrence of the unpleasant gastric sensations that I used to have.

2. Neither the pathologist nor any of the surgeons at the Manchester Royal Infirmary have seen a single case of ulcer occurring at the site of the stoma (gastro-jejunal ulcer) nor have they seen a jejunal ulcer. The only case of ulceration occurring in this region was an acute ulcerative process that almost separated the jejunum from the stomach, but this was four years after the operation had been performed.

contractions of various parts of the stomach. The spasm produces a narrowing of the lumen that is of great functional importance, causing a definite obstruction to the passage of food. By an indiscretion of diet or want of mastication something too large to pass easily through the channel has to be forced through by powerful peristalsis. This leads to an abrasion at the point where the lumen is narrowed and where there is the constant irritation of food passing over it. Also a surface is exposed and is not structurally fitted to withstand the action of the gastric juice. In this way an ulcer is formed which, in its turn, perpetuates the original spasm that determined the site of the ulcer. A vicious circle is established, the ulcer now being actually the cause of the spasm that prevents healing, so that even if the original cause of the spasm is removed there may be little, if any, improvement in the local condition.

The stomach and duodenum are, as it were, the storm centre for the alimentary tract to which peripheral stimuli are referred, the result being either a general irritable condition of the stomach and duodenum, or spasmodic contractions of one or more parts of the stomach. These stimuli are referred to the stomach and duodenum either directly, from swallowed septic matter, or indirectly from lower parts of the tracts as in the case of lesions of the small intestine, as indicated radiographically and proved by operation; as in the mucous colitis cases and constipation cases as indicated radiographically and also by the subsequent history of the cases.

Appendix dyspepsia is too well known to need mention. Radiographically it is indicated as a rule by duodenal irritation and Moynihan tells of the very large percentage of duodenal ulcer cases in which there is evidence of old appendicular trouble.

Now these spasmodic contractions give rise to all the radiographic appearances of actual lesions—on more than one occasion a surgeon has re-opened an abdomen, at my suggestion, because of the persistent hour-glass contraction that has been noted above the stoma of a gastro-enterostomy which was functioning perfectly. Three of these patients were actually observed vomiting from the upper sac of the hour-glass con-

traction, the pain and tendency to vomit disappearing when once the food had passed down into the lower sac, and yet no cause was found at the operation to account for the contraction. In several cases where I have reported definite pyloric obstruction the surgeon could find nothing to indicate gastro-enterostomy, and in two of them a subsequent operation showed thickening of the pylorus. There are many cases of various types, all pointing in no uncertain manner to the obvious conclusion that the spasmodic contraction is of as great importance, functionally, as the organic, and that these apparently purely spasmodic contractions may be replaced by definite organic lesions has been actually noted in some two or three cases in which a second operation has been undertaken.

That ulcerations of the alimentary tract are irritable and give rise to spasmodic contractions I have seen proved time and again, *e.g.* ulcers of the œsophagus that only caused symptoms, and obstruction, after the patient took dry bread crumbs. (The ulcers were seen through the œsophagoscope afterwards.) The same has been noted in the stomach on several occasions. Hour-glass contractions with retention of food in the upper sac have often been found, at operation, to be due to quite small ulcers with practically no cicatrization.

If my contention is correct (that purely spasmodic contractions are capable of producing functional obstruction which leads to trauma of the mucous membrane, and that this ulcer or abrasion is itself capable of producing a similar spasmodic contraction), it follows that, when once an ulcer is formed, it will perpetuate the spasmodic contraction even if the primary cause of the spasm is removed. Hence it is clear that removing the primary cause will not necessarily cure the patient, for the ulcer, when once formed, continues to induce a spasm that tends to the perpetuation of the ulcer. If, on the other hand, an ulcer or abrasion has not occurred, the removal of the primary cause, whether it be teeth, nasal discharge or swallowed pulmonary discharge, stagnation of fæces, appendix inflammation or some other cause, will probably bring about a rapid and lasting cure of the patient. It is probable therefore that the chief factor in the production of gastric ulcer is a physical one, but the connection between the various

primary conditions and the spasmodic contractions they produce is a problem that is beyond the reach of radiography.

This theory covers the whole of the many and varied observations I have detailed and I believe accounts satisfactorily for them all, including the failure to produce gastric ulcers artificially that are comparable to those met with in man. To what extent the gastric juice is responsible I cannot say, but it seems reasonable to suppose that when once an abrasion is formed, a surface is exposed that is not adapted to withstand the action of the gastric juice and will therefore tend towards the extension of the ulceration.

DUODENAL ULCERS.

In the duodenum the conditions leading to the formation of ulcers, do not appear to be the same as in the stomach. Observations show that in every case of duodenal ulcer, and also where there is duodenal irritation, the stomach begins to empty itself very rapidly and one can easily see quite large shadows pass through the duodenum. In some cases there is a separate bolus persistently present in some part, as if a pocket was formed, but in spite of large quantities passing through, I have very seldom observed obstruction. Whenever this has been seen, there has been definite cicatrization or some external cause found at the operation to account for the retention. It seems therefore as if spasm, as seen in the stomach, is not associated in the same way with duodenal ulceration and I think it likely that some other influence is at work in these cases. The conditions that obtain in the stomach and duodenum during life are as yet only partly understood, and the various factors that control the passage of the food through the pylorus are by no means clear. I have given my reasons (p. 52) for believing that the control of the pylorus is influenced by the duodenal condition. In all cases of duodenal ulcer and of duodenal irritation one sees very abnormal pyloric relaxation was evidenced by the passage of large masses of food, whereas in the normal subject it is practically impossible to see the food passing through the duodenum, so fine is the stream, and so rapid the segmentation and shredding of the food. The appearances suggest that the segmentation con-

tractions that normally shred the food in the small intestine are absent in the duodenum when there is irritation or ulceration, otherwise the large shadows would not be seen in the duodenum. We know, therefore, that the pylorus is abnormally relaxed to these cases and one suspects that the relaxation extends to the duodenum and that the relaxation is sufficient to counterbalance whatever tendency towards spasmodic contraction the ulceration might give rise to. The absence of a spasmodic contraction in these cases would account for the fact that they frequently heal spontaneously, but the causation of these ulcers does not appear to be the same as in gastric ulcer.

My impression is that the majority of duodenal ulcers are due to trophic changes of a reflex nature and dependent in most instances on deferred stimuli arising from lesions or toxæmias of lower parts of the alimentary tract. The most fruitful source of such stimuli is probably in the terminal ileum and often dependent on the appendix in some way.

There are also certain cases of duodenal ulcer that are associated with gastropptosis. In these it is nearly always noted that the right kidney is also dropped considerably, and one cannot help thinking that there must be a definite connection between nephropptosis and duodenal ulcer in these cases, for one has seen the gastric symptoms disappear completely as the result of nephropexy.

CHAPTER XII.

THE LARGE INTESTINE.

To write a paper on the *x*-ray examination of the large intestine would have been comparatively simple a few years ago. One would have been content to take the anatomical structure and have dealt with it according to the circumscribed views that we inherited. To-day it is different, for we know that the large intestine is but one part of a complex organisation, so closely linked together that it is almost impossible to separate out the constituent anatomical parts and deal with them separately. The conception of "water-tight" compartments with anatomical boundaries in the alimentary canal is disappearing as more and more evidence accumulates to show the interdependence of the whole alimentary tract. Here and there we get glimpses of reflex spasms, *e.g.*, a pyloric spasm from some apparently slight abnormality in the ileo-cæcal region, that indicate a network of nerve centres, relays and sub-centres that are as yet beyond our ken.

The physics of osmosis, a long line of test tube experiments with secretions and ferments, a vague and quite unessential idea of some movement of the stomach and intestine, used to fill the whole field of our conceptions of digestion. Perforce we had to be content with it. In recent years it has become possible to study the mechanics of digestion and the movements of the food through the tract, and it has become more and more evident that the conditions with which the surgeon is called upon to deal are those that are associated with faulty *mechanism* rather than faulty *digestion*, with the result that there is perhaps a tendency to forget that there is a chemistry, as well as a mechanism, of digestion. It is the mechanical factor of digestion that is to-day passing under review; the *fons et origo* of irregular muscular contraction is being sought, and the investigation shows very clearly that the whole musculature of the alimentary

tract is linked up by a nervous system that is probably as complex as that of the brain itself; a system, possibly subservient to the central nervous system, but also capable of automatically exercising a selective action on ingesta, of calling secretory glands into activity, of preparing the tract for the food that is coming (*e.g.*, emptying the ileum when food is taken into the stomach), and many other functions that we hardly suspect as yet. Verily there are "more things than are dreamed of in your philosophy." "What a wonderful thing is man!" I know of no subject that opens out a wider field for investigation than this, or one that calls for more careful and accurate observations. It bristles with interlocked problems, some of which have been answered in a dogmatic and quite unscientific spirit by various workers. If I fail in dogmatism—and usefulness possibly—it is because I feel so deeply the responsibility of attacking problems of such vital importance, in anything but the most open frame of mind. The profound complexity of the whole mechanism with its wonderful efficiency, is a thought that should deter any surgeon from mutilation. But there are those who know better than the Great Architect, who call the large intestine by names that suggest that, if they had the making of man, they would do better. If I cannot go with these it is because I believe that the Great Architect turned out men fit to live in this world; whose cæca, although apparently insanitary, are yet fulfilling the functions they were intended to perform. That certain wholesale removals are successful is no proof that they were necessary, but rather that somewhere in the large mass removed lay the cause of the trouble. Some day, these large operations will be unnecessary, but that day is not until we can place a finger on the exact spot where the trouble arises. In the meantime one is astonished, not at the brilliance of the surgery displayed, but by the tolerance of the human body.

What is one man's food is another man's poison, is an old saying, and expresses the fact that this complex nervous mechanism controlling the digestive tract is not standardized; that each individual is a separate creation, each bearing outward resemblance of a standard article. Even the gross appearances of the normal stomach vary in such a way that it

is only a study of a large number that will give a true conception of the variations that are possible within the limits of the normal. And, if such is the case in the stomach, it is even more so in the large intestine, and one is driven to accept wide variations from standard as being quite normal because function is fulfilled. In fact the only real test of abnormality that we can apply is that of functional disability : when one has seen such extreme cases as those women who are perfectly healthy and yet defecate but twice or thrice a month, one is not justified in dogmatising on the subject of stasis in the large bowel. We have inherited the theory that it is normal to defecate once a day, and if so, all is well ; if not, the anxious mother gives purgatives, and attempts to establish this standard. Nobody is more alive to the importance of keeping the bowels open than I am, but each person is an individual, and it may not be possible for the individual to acquire a twenty-four hour standard for defecation ; the persistent use of purgatives in such cases is likely in time to lead to real troubles. One sees so many instances of women in whom the opaque meal takes forty-eight hours or more to reach the rectum, in spite of the fact that the whole tract has been cleared out in preparation, that one believes that in many of these the natural habit would be forty-eight hours or more. We are so obsessed with this twenty-four hours' idea that, willy nilly, our children are moulded to it, and undoubtedly it is right for the large majority, but my point is, that there may be very wide variations within the normal limits. If we could accept a universal twenty-four hours' standard, the difficulties of writing on this subject would be comparatively slight and dogmatism could be indulged, but, since each case must be treated as an individual, the evidence deduced from watching the passage of a standard meal must be most carefully balanced with the clinical picture before deductions are made.

It has been shown that the food passes into the cæcum in part by pressure from behind, *i.e.*, that the ileo-cæcal valve acts as a sphincter and that its function is not only to prevent regurgitation but to regulate the flow of chyme into the cæcum. Ileal stasis is, up to a point, a physiological condition and collections of shadows should be found in the last

coils of the ileum in about three hours from the time the food is taken, while the coils should be approximately empty in eight hours. These figures are necessarily very approximate, for the variations in the rate of emptying of the stomach and the passage of food through the small intestine vary within wide limits. For instance, to take two extremes, in duodenal irritation one has seen shadows arriving at the ileo-cæcal valve in well under half an hour, whereas in delayed emptying of the stomach, it may be an indefinite time before the food reaches this point. It is therefore very difficult to say what constitutes ileal stasis; not only have we this factor to consider but also the knowledge that accumulations of fæces in the cæcum will give rise to back pressure, and in every case of constipation, when the cæcum is loaded, one will see evidence of ileal stasis. But this form of stasis is readily dissipated by means of an enema and a purgative. Many such cases have been labelled ileal stasis and published as such in the journals. On enquiry one finds that these patients have not been prepared and that no preparation is undertaken *because* no ileal stasis is found after the bowels have been cleared. Surely if this is the case the symptoms cannot be due to ileal stasis, or a simple purgative would invariably relieve the condition and cure the symptoms. It is therefore necessary that all patients should be carefully prepared for examination, for, if we fail to do so, we will discover a stasis that is, in reality, nothing but back pressure from cæcal constipation, while on the other hand, if purgatives are still exerting their influence, there will be a tendency to obliterate any ileal stasis that may be present. The routine I myself adopt is to give a purgative thirty-six hours before and an enema on the morning of the giving of the first opaque meal. After this, apart from the giving of the opaque meal, the patient leads as nearly as possible his ordinary daily life and observations are made from time to time according to circumstances. When the time available for the work is divided by hospital and private practice, one has to adopt some definite routine for "double" feeding and, in my own case, I have the patient prepared as stated and, at the first examination, watch the filling of the stomach, and the manner

in which it empties itself. Instructions are then given for feeding on the following day at such an interval before the second examination that one will expect to find the greater part of this second meal in the cæcum. This will vary from three to seven hours according to the rate of emptying of the stomach observed at the first examination. When the patient is again under observation one should find the whole of the food given at the previous examination in the colon, the head of the shadow being somewhere in the region of the splenic flexure. (When the shadow has passed well beyond this point it is usual for the patient to have had a desire to defecate or to have actually passed a motion.) The food given say, seven hours before, should be in the cæcum and ascending colon with a comparatively small amount in the last coils of the ileum.

In my opinion it is advisable to use as little of the opaque mixture as is compatible with obtaining clear definition in the picture and also that the excipient should be a *real food*. Bread and milk into which the barium sulphate is mixed is what I employ, and it gives good results. Suspensions of opaque salts are, to my mind, to be avoided for the reason that they introduce a possible source of error quite needlessly. The same applies to the massive doses, six or eight ounces of opaque salts, that are sometimes given. Two or three ounces with each of the opaque meals is ample.

Anatomy.

The anatomy of the terminal ileum calls for comment: the last four inches look as if they were different in structure, and function possibly, to the rest of the ileum. This portion does not pursue a tortuous course but runs more or less straight upwards and outwards to the cæcum. Its calibre is smaller, and the chyme forms a continuous shadow which is quite different from the other parts of the small intestine. It looks as if the circular fibres were more evenly developed and that tonic action was a more persistent feature of the muscular action. One believes that the whole of this terminal portion is closely associated with the sphincteric and valvular action that is ascribed to the ileo-cæcal valve itself.

The anatomy of the large intestine, as shown radioscopi-

cally, gives a picture of a more or less uniform tubular organ with regular haustral segmentation, from the cæcum to the rectum. One sees no evidence of any sphincter between the cæcum and ascending colon, in fact one has developed the habit of thinking of the cæcum and ascending colon as one organ. For whenever one sees any tendency to distension and "sloppiness," it seems to involve the whole organ as far as the hepatic flexure.

The hepatic flexure is much lower than one expects, it hardly reaches the level of the umbilicus when the patient is standing, and frequently gives the impression of being prolapsed, but the splenic flexure always ascends to a very high level into the dome of the diaphragm, the actual angle often containing a collection of air that is as closely related to the diaphragm as the fundus of the stomach.

Both flexures look at first sight as if they were acute angles in the gut, but this is not so: it is due to fore-shortening of the two limbs of a U-shaped bend.

The transverse colon is far from horizontal. Its general direction is at an angle of 45° up to the splenic region but there is almost invariably a loop, that drops down to a much lower level.

The descending colon drops straight from the splenic flexure to the iliac crest and at this point becomes fixed by the peritoneum. In a proportion of cases the descending colon drops down over this fixed point and there is a tendency to kinking. This, I believe, is the only point at which "kinking," apart from adhesions, occurs.

The only point about the anatomy of the sigmoid that strikes one, is the close relationship that the loaded cæcum and sigmoid may bear to each other. Sometimes they seem to be actually in contact.

Physiology.

The radiographic indications of the physiology of the large intestine suggest a series of problems. The first of these is in the terminal ileum. Someone, Moynihan I believe, made an epigram to the effect that the most frequent seat of a gastric ulcer is in the right iliac fossa. All surgeons must now appreciate the underlying truth of this. Little by little facts

are coming to light that indicate a very close connection between the ileo-cæcal region and the duodenum. Apart from the surgical evidence as the result of operations on appendices, some of them apparently healthy, we have radiographic indications that taking food into the stomach tends to make the ileum empty into the cæcum (p. 54), and now I have a series of cases that indicate another type of connection, *i.e.*, a definite ileo-pyloric reflex—back from the terminal ileum to the stomach:—

In certain cases, duodenal irritation or ulcer type, the stomach begins to empty very rapidly, overloading the small intestine. In a short time (say $\frac{3}{4}$ hour), with a small quantity of food, the stomach is empty. And yet when a reasonably large meal was given it was found that, instead of being empty in the usual four hours, there was actual delayed emptying, and in some cases fully a half was still present after seven or eight hours. On again watching the progress of the food one noticed that it was, so far as one could determine, when the shadows reached the terminal ileum that this rapid emptying and general activity of the stomach ceased, and in each case there was quite well defined ileal stasis, and very little food seemed to be passing on into the cæcum. These observations give the impression that the terminal ileum is abnormal in some way, and when once the food gets down to this point it causes a reflex closure of the pylorus and quieting down of the gastric activity, *i.e.*, a message from a nervous centre in the ileo-cæcal region to the centre in the duodeno-pyloric region to the effect that as much food had come down as can be dealt with, and a request to shut off supplies. In nearly all these cases one found, at a seven hours' examination, that there was a complete gap between the food in the stomach and that which was collected in a mass behind the ileo-cæcal valve. Those cases that have been operated on have all shown definite evidence of old inflammatory changes in the ileo-cæcal region. (This subject is more fully dealt with at pp. 53 and 98.)

The importance of this observation is obvious, and throws new light on that curious manifestation, appendix dyspepsia.

Lane's kink I have not recognised radiographically, and I am far from convinced that it is a cause of either ileal stasis or of this ileo-pyloric reflex.

The ileo-cæcal valve has been very carefully studied by Case, Hertz, Cole and others by means of opaque enemata, and, apparently, it should normally be quite resistant to the injection. No leakage should take place into the ileum. In a certain proportion of cases (16 per cent.),¹ however, it is found that the injection does pass through to some extent, *i.e.*, the valvular action is incompetent. In one of my own cases, I saw an injection flow quite freely, not only into the ileum, but also in the jejunum; some of it I actually located in the duodenum.² Case states that when this phenomenon is noted radioscopically, the incompetence can be confirmed at the operation by "milking" the food or air in either direction, and Kellog³ has devised an operation for the repair or the formation of a new valve. As a cause of ileal stasis this defect is well worthy of consideration, and Cole asks if it is likely that the small intestine will tolerate regurgitated fæcal matter without giving rise to symptoms of some kind, probably referred to the stomach. This incompetency of the ileo-cæcal valve is probably the explanation of the fact that patients sometimes state that they can *taste* a soap enema. One patient is stated to have actually vomited a part of an oil enema within half an hour.⁴

The *appendix* is readily recognised when it is filled with opaque food, and, in a systematic examination, one always looks for it with the patient lying down. By using a wooden kitchen spoon, as suggested by Holzknicht, the shadows can be manipulated and the cæcum can be pushed up out of the pelvis, as described by Case, so that the lower end of the organ is clear away from the shadows of the ileum. If the appendix is filled it is clearly seen, and in one case I had the opportunity

1. J. T. Case xviith Interna. Congress, 1913.

2. This valve was found to be perfectly competent at the operation and no cause for the phenomenon was discovered. In fact, the only abnormality noted was a band of adhesions slightly constricting the transverse colon.

3. Surgery of the ileo-cæcal valve. Surg., Gyn. & Obst., 1913.

4. Cantani, *Virchow's Jahresbericht*, 1879. Bd. ii, p. 180.

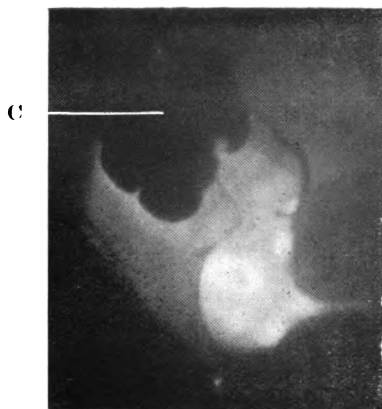


Fig. 64.

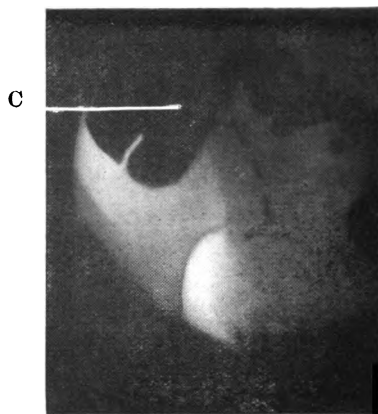


Fig. 65.

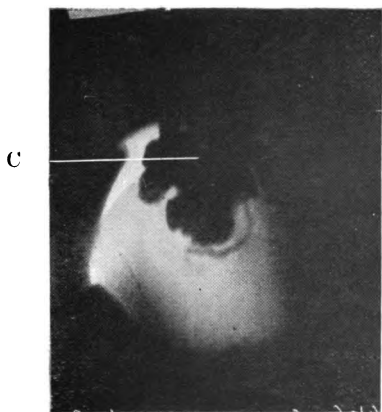


Fig. 66.

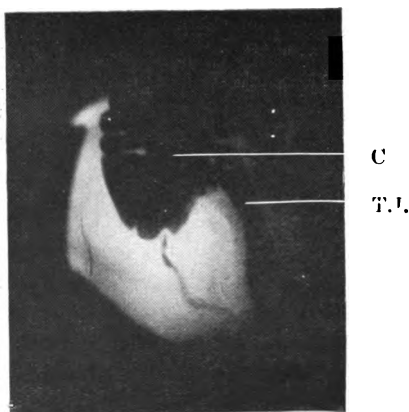


Fig. 67.

Fig. 64. Appendix. Cæcum (C.).

Fig. 65. Same case shortly after, showing the shadow cut up into beads. Case illustrating movements of the appendix (p. 125).

Fig. 66. Appendix adherent by mesentery to cæcum; the tip lies free.

Fig. 67. Long appendix lying free. (C.) Cæcum. (T.I.) Terminal ileum.

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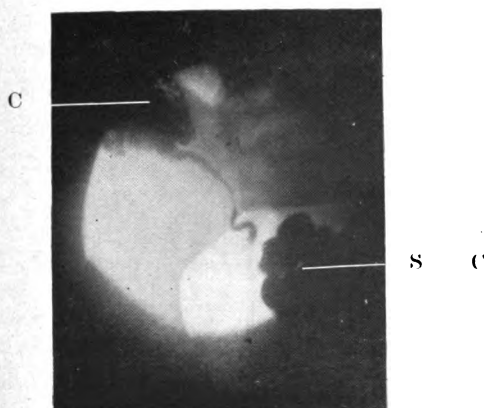


Fig. 68.

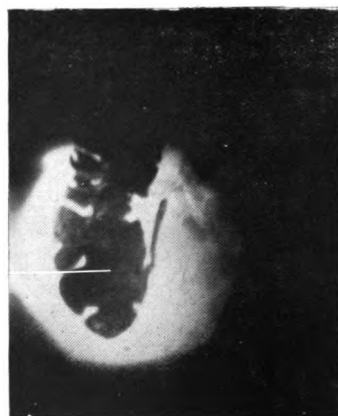


Fig. 69.

Fig. 68. Long appendix with tip fixed to sigmoid. (C.) Cæcum. (S.) Sigmoid.

Fig. 69. Appendix. Tip fixed to terminal ileum. Adhesions about appendix and duodenal ulcer.

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of watching the various movements that can take place. It was a very long appendix, nearly six inches at the operation, and it was completely filled. I saw the shadow cut up into five oval beads and then the organ emptied itself. A few minutes later the shadow again appeared and remained stationary with the exception of a worm-like writhing movement while the food canalised it, which persisted for a few minutes after it was completely filled. In my cases, in hospital, I find the appendix in about 40 per cent. of the cases only. Case finds the organ in about 50 per cent. of all cases, while George, of New York, gives his figures as high as 80 per cent. Case states that he can sometimes "milk" the food into the empty appendix.

By manipulation one can tell whether it is adherent or not and also whether it has a mesentery. Those appendices that point downwards are usually lying free, and are not bound down, while those that point in other directions are often adherent to some other viscus. Apart from making a negative diagnosis when one sees the appendix functioning and lying free, or from detecting adhesions, I have little confidence in making deductions from the appearances as to the presence of trouble caused by the appendix. Leaving out acute cases, my impression is that the effects of appendix trouble are associated rather with referred irregularities of the terminal ileum than with the actual appendix itself, and that in every case where the appendix is removed it is most important to search out and divide any small adhesions there may be, no matter how insignificant they may appear.

Movements of the Large Intestine.

From the time when x-rays first showed the bismuth mixed faeces, it was evident that the old theories as to the movements of the large intestine were not correct. The extraordinary thing was that no movement of any kind was seen, and yet, from time to time, one noted that the head of the column had passed through many inches of the colon between two examinations, separated only by a few minutes. Holzkecht¹ was the first to see and describe such an actual movement of

1. Münch. Med. Woch., 1909, No. 47.

the faeces as would explain this fact; it was apparently a sudden movement, *en masse*, of the whole column in the colon, and took place in some three seconds. He recorded two observations of the phenomenon out of a large experience, and stated that he believed this to be the normal mode of progress through the large intestine. No further observations, or comments on this observation, were made, and it was not till nearly two years later that the writer¹ saw one case and happened to obtain radiograms of another as the movement was taking place. These observations were published, together with the radiograms, but it was not until Hertz² studied the phenomenon systematically that this somewhat revolutionary theory became more or less established. Taking advantage of the fact that the call to stool usually follows close after a meal, the gastro-colic reflex, he made a series of observations on medical students and confirmed the Holzkmnecht theory. He saw the movement actually taking place in three of the patients so examined. In all, I have seen this movement take place some nine times, and am absolutely convinced that it is the natural and normal movement. In three students whom we examined as they ate their breakfast and for a time afterwards, Mr. J. Morley and I saw the movement in one case most perfectly, and, in another instance, we happened to look at the patient just as the movement was about over. Jordan has also recorded observations on these movements; but Case's report before the XVIIth International Congress of Medicine is the most extensive and scientific treatise on personal observations that I have seen.³

There is not, so far as I know, any theory, except this of mass movement, that will explain the facts as we see them. All the observers agree that, without subjective sensations of any kind, the haustral segmentation disappears and the whole mass rushes suddenly, in three seconds Holzkmnecht suggested, through a length of colon. In all my cases it happened that the head of the column was in the region of the hepatic flexure and when the movement finished the head of the shadow had

1. Arch. Roentg. Ray, 1912.

2. Journal of Physiology, Oct. 17, 1913.

3. J. T. Case, X-ray observations on colonic peristalsis, etc.

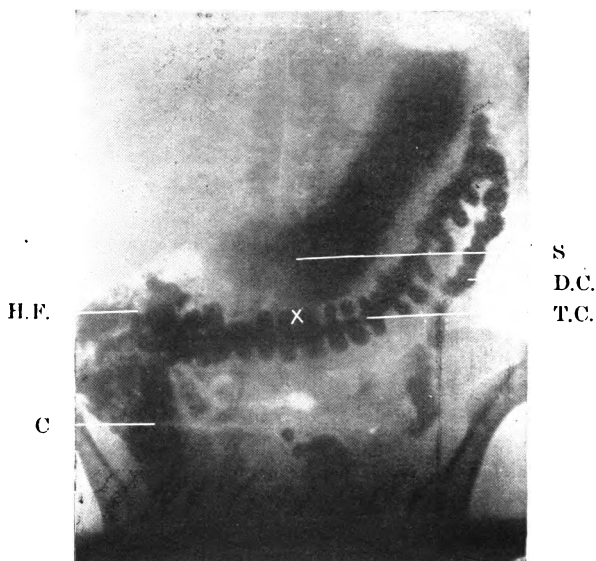


Fig. 70.

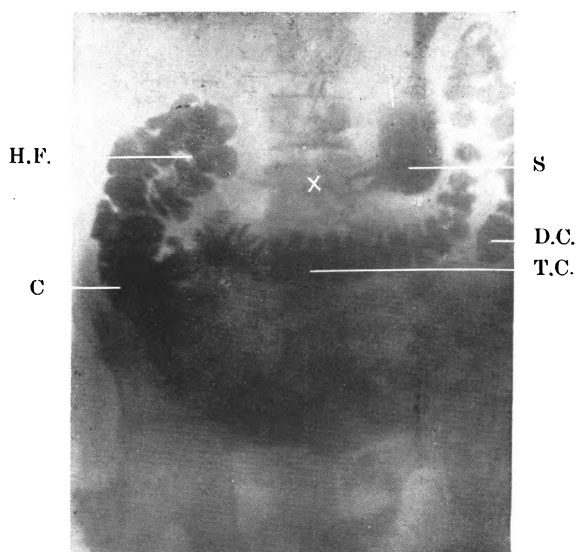


Fig. 71.

Fig. 70. Shows relation of colon to stomach. Note the hepatic flexure is ptosed while the splenic flexure extends nearly to the diaphragm. (S.) Stomach. (C.) Cæcum. (T.C.) Transverse colon. (D.C.) Descending colon. (H.F.) Hepatic flexure.

Fig. 71. Loop of transverse colon adherent to the cæcum. (S.) Stomach. (C.) Cæcum. (H.F.) Hepatic flexure. (T.C.) Transverse colon. (D.C.) Descending colon.

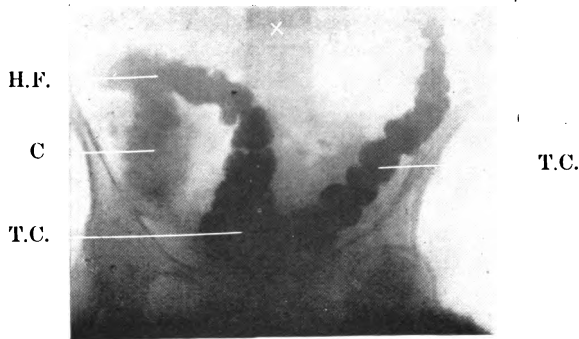


Fig. 72.

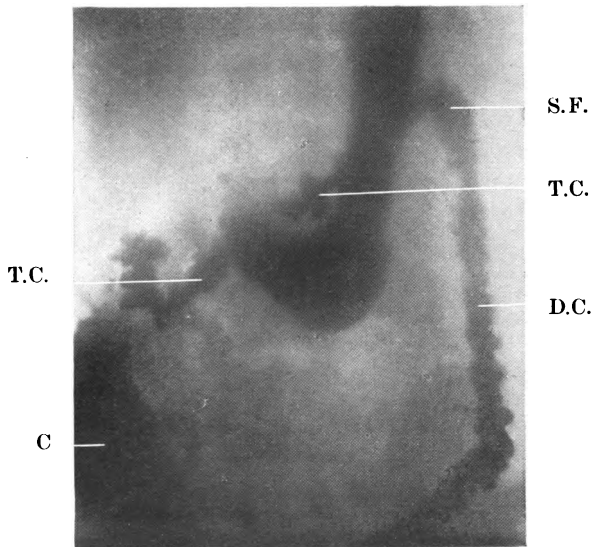


Fig. 73.

Fig. 72. Marked loop of transverse colon adherent in pelvis. Appendix adhesions.

Fig. 73. Displacement upwards of transverse colon so that it lies across the stomach. No cause for this found at the operation but in another similar case an old ruptured ovarian cyst was responsible for adhesions anchoring the colon in this position.

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passed on to the splenic flexure or further.¹ The whole process is over in a very short time, and the haustral segmentation, and general picture of still life, are almost immediately restored. My impression is that the *perfectly* natural movement is somewhat slower, at any rate it has appeared to be so since I had my patients prepared by purgatives thirty-six hours before the first meal, *i.e.*, sixty hours before one expects to observe the large bowel. In one instance I timed the passage from the middle of the transverse colon to the pelvic brim as fifteen seconds, but in all the other cases the passage was so unexpected that it was almost completed before one had time to realise what was happening. The mechanism appears to be, in the first place, a relaxation of the tonic action of the muscular coats followed by a big peristaltic wave that sweeps the whole contents along. This movement probably occurs some three or four times a day.

Incidentally it is interesting to note that the firm and solid appearance of the colon shadows both on the plate and also on palpation, is due to the tonic action holding the more or less fluid fæces in definite form.

Constipation.

I see no alternative but that we accept this *en masse* theory of the movement of the food through the large intestine, and, such being the case, we must revise all our conceptions, not only of the normal but also of the pathological. The movements certainly tend to occur after a meal, quite shortly after it is eaten—Hertz's and my own observations were all made at breakfast time, as being the most likely, and we were both successful in seeing the phenomenon, while in ordinary routine work one does not see evidence of movement during the examination in more than a very small percentage of cases. In private practice, in the afternoon, I have only once seen

1. I have recently seen a case in which a portion of the shadow was cut off from the ascending colon and traversed the whole length of the colon into the sigmoid. When the movement was over, a slight antiperistaltic mass movement took place, in the nature of a rebound, and part of the shadow again returned through the iliac colon to just above the iliac crest. The whole movement took about 10 seconds.

any of the movements and comparatively seldom noted that any movement has taken place between the observations. The call to stool does not necessarily follow one of these movements, in fact it has been entirely absent although in two of the cases the rectum was already full.

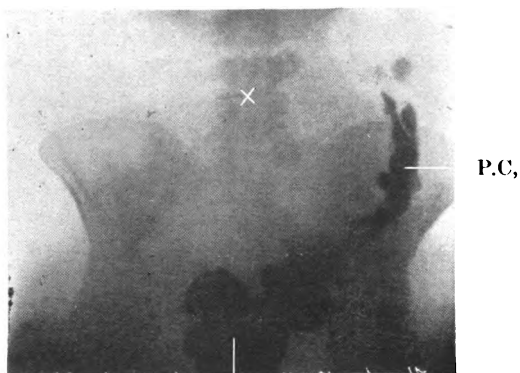
From his observations on students, Hertz¹ suggested that the average normal times in which food should reach various points were: cæcum $4\frac{1}{2}$ hours, hepatic flexure $6\frac{1}{2}$ hours, splenic flexure 9 hours, brim of pelvis 11 hours. He is insistent on the fact that these are only average times and wide differences are possible even in perfectly normal subjects. If we accept this "mass" movement, occurring some three or four times a day, as the normal, it is evident why these wide variations are possible. Small eaters frequently suffer from constipation while big eaters usually have little trouble in this direction. It seems likely therefore that a big meal produces a stronger gastro-colic reflex than a small one. Many people find that a glass of warm water before breakfast helps to produce a post-prandial motion, and one would suggest that the water helps to clear out any collections left in the ileum and to fill the lower part of the cæcum so that when the normal gastro-colic reflex of the breakfast table acts, the food is already well into the large intestine and ready to respond to the stimulus.

Constipation has been defined by Hertz and others, but, holding the views I do, I would rather leave out any definition, for each case is an individual and this must not be forgotten. One sees patients who are always out of health if a daily action is not obtained, while one has also seen health regained when patients have ceased to worry the intestines into daily actions and reverted to a habit of twice a week. My own experience in the study of these cases indicates that there are two and only two real seats of stagnation in the large intestine, apart from kinks and adhesions, *i.e.*, the cæcum and the rectum. Of these the rectal stagnation is the most frequent, and to it Hertz has given the name of dyschesia. It is in reality a fault of defecation rather than a true constipation. In these subjects it is extraordinary to see the way in which the

1. Constipation and allied Intestinal Disorders. London, 1909.



Fig. 74.



R
Fig. 75.

Fig. 74. Dilated rectal ampulla in a case of dyschesia.

Fig. 75. Dyschesia—72 hours after food, rectum full but no call to stool. (R.) Rectum. (P.C.) Pelvic colon.

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shadows accumulate and form great masses in the rectal ampulla without any call to stool being felt. The rectum is ballooned out to a great size and is, presumably, atonic, and when the bowels are moved one finds that it is only a part of this shadow that has disappeared.

But in constipation proper, the delay is nearly always in the cæcum and ascending colon, and these portions remain filled for days at a time, in spite of the fact that some of the contents may have moved on to various portions of the colon, *i.e.*, it is a defect in this "mass" movement, a defect not of one part of the colon, but of the organ as a whole. The position of the head of the shadow is not necessarily an index to the site of the portion of the gut that is responsible for the delay. One believes rather, that the constipation is more likely due to some fault in obtaining a more or less fixed point,* or a temporary sphincter at some point, from which to work, than to a defect in the movement itself. One has never seen, nor are there any records to show, that this movement takes place in the cæcum itself. One has often seen movements in the lower part of the cæcum of a peristaltic nature and it is possible that these should be sufficient to raise the food to a position in the ascending colon in which this "mass" contraction can catch hold and carry the food along. As the result of his researches in comparative anatomy Keith believes that there is some form of sphincter between the cæcum and ascending colon. Possibly, although one sees no radiographic indications of it in the ordinary course of events, there is some sphincter as he suggests, and its function may be to prevent regurgitation when this "mass" movement is in progress. In support of this hypothesis there are several observations: one has never seen the cæcum empty suddenly between two observations, nor has this "mass" movement of the shadow been observed to clear out the cæcum. On the other hand one has frequently observed large shadows left in the cæcum while the contents of the hepatic flexure and transverse colon have passed far on towards the pelvic colon. Moreover, although the large intestine is always swept *clean* (except for definite scobola possibly), there are often traces of the opaque meal left in the cæcum for days, *i.e.*, it is not swept clean.

* A "point d'appui."

This mass theory is so unparalleled in the human body that one hesitates to go any further than this brief suggestion of the normal: one finds widely ballooned colons and greatly narrowed colons, *i.e.*, apparently atonic and hypertonic types, and these have been described as the causes of constipation, but I doubt whether they are. Rather, I would suggest, they indicate the conditions of the colon *between* the movements, for I happen to have seen the "mass" movement sweep the shadow along both types of colon, and in both the movement was perfectly effective in displacing the contents. The part played by the cæcum is not clear but, if my deductions are correct, this portion of the large gut has a separate mechanism or peristalsis for mixing the contents and feeding them into the ascending colon, preparatory to the occurrence of the "mass" movement. (See further note on p. 132.)

The subject is full of difficulty; not only are the observations few and far between, in spite of the vast number of patients examined, but also because the whole new conception is revolutionary and alien to the traditional line of thought—it would be easier if we had not been brought up on arm-chair conceptions of what takes place, for unconsciously we attempt to make our observations fit in with our preconceived notions.

Antiperistalsis is also stated to occur in the large intestine, in fact Case gives incontrovertible proof of it, and it is likely that it can occur in the normal healthy subject. In fact, to-day, I have actually seen it occur in a small way after a "mass" movement had taken place. It is probable that this movement is exactly parallel to the usual forward "mass" movement but occurring in the opposite direction. One does not see any such movement on injecting an enema, but even a pint will be carried back to the cæcum in most cases.

Besides the "mass" movement, one sees, from time to time, changes in the shape of the haustral sections of the large intestine, especially in the distal colon. It is not at all a marked movement and has probably no connection with the progress of food along the intestine. Presumably it is connected with digestion and acts by churning.

Rieder described "large pendulum" movements which appear to be much the same as the smaller haustral churning

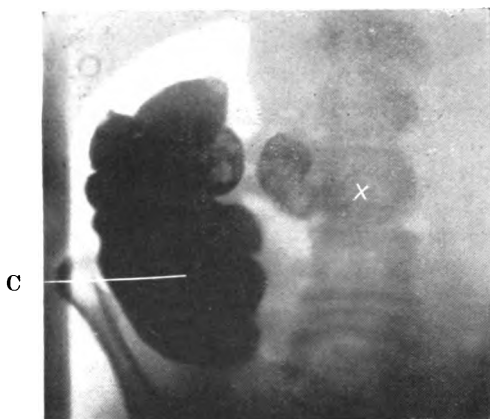


Fig. 76.

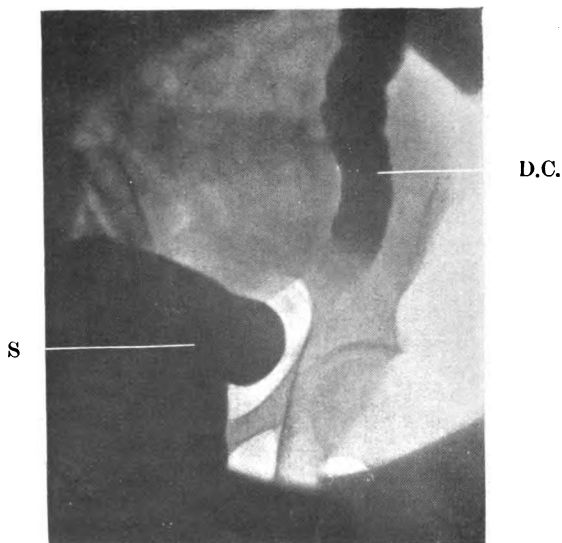


Fig. 77.

Fig. 76. Dilated caecum in a case of neoplasm of the transverse colon.

Fig. 77. Carcinoma of the sigmoid and pelvic colon. Barium enema injected and distending the rectum and sigmoid (S.). Very slowly it filled the descending colon (D.C.) and one could just palpate a resistance at the site of the gap in the shadow and detect that this coincided with the break in the continuity of the inflowing stream of opaque enema. The gap in this case is much more marked than usual.

of which we frequently obtain evidence. Case states that in every instance that he has seen of the larger movement, it has been the precursor of a large mass movement.

Neoplasms.

Turning now briefly to the organic lesions of the large bowel one finds that it is only when there is obstruction of the ascending and first part of the transverse colon that one can obtain definite evidence from feeding the patient. In these cases one gets great ballooning of the cæcum and ascending colon. But in the case of growths or other obstructions occurring beyond this region, there is only one method of investigation in early cases, and this is by means of the opaque enema delivered from a glass tunnel dish through a tube. Watching the course of the opaque injection we can see where it is obstructed and where the stream is narrowed, and with the aid of palpation we can as a rule form a very good idea as to the nature and extent of the lesion that is causing the trouble. In this work, as in the opaque meal work, it is very important to confirm observations by re-examination, for spasmodic contractions may be most misleading. Especially is this the case with the upper sigmoid, and on one or two occasions I have been misled. When an obstruction is noted at the pelvic brim I have found it advisable to lay the patient on his face, a proceeding that sometimes allows the injection to pass on quite freely.

In advanced cases, however, one nearly always finds collections of air and liquid in the distended bowel behind the obstruction. But, as already suggested, it is only in advanced cases that this ballooning of the bowel occurs. It is in this type of case that evidence of antiperistalsis is most often noted.*

* J. T. Case gives the following summary of the points on which he bases his diagnosis in carcinoma of the large bowel (*Lancet Clinic*, Feb. 21, 1914):—

1. Exaggeration of the normal antiperistalsis, giving the appearance of "peristaltic unrest" to the bismuth content above the site of the obstruction.
2. Arrest or hindrance in the onward progress of the bismuth meal.

It is interesting and instructive to note that the food given by the mouth seldom forms heavy shadows behind an organic lesion in the distal large intestine until the obstruction is very far advanced, in fact the head of the shadow seldom reaches the obstruction, and more often than not the shadow is comparatively thin—as if the colon was hypertonic in its efforts to overcome the obstruction, and it is only when this hypertonic state gives way that the gut behind the obstruction becomes distended and filled with faeces.

Palpation of the loaded colon will give the clue to the presence of Jackson's membrane, adhesions, kinks and other abnormalities that occur, but of these I have not time to write at present.

Further Note on Constipation.

Since writing the above, I have on two occasions (1994, 2022) noted the formation of a definite constriction, a "point d'appui," such as I suspected was necessary for the efficient action of the mass movement. In each case it was near the hepatic flexure and was not evident until this was palpated out with the spoon. I am not absolutely certain on the point, but believe that it was formed after palpation of the cæcum in

3. Arrest or noticeable hindrance in the ascent of the bismuth stream when giving a bismuth enema.

4. Coincidence of a palpable tumour with the point of hindrance in the progress of the bismuth meal or the bismuth enema.

5. A filling defect in the shadow of the bismuth-filled colon. Frequently the filling defect is digitated, indicating a cauliflower growth. At times, it may be annular so that one may diagnose an annular carcinoma.

6. The amount of bismuth enema which may be injected is often indicative of the site of the lesion.

7. The colon is often markedly distended by gas, and gas collections are seen surging backward and forward, due to the alternations of peristalsis and antiperistalsis.

8. Marked ileal stasis when the neoplasm involves the cæcum, ileo-cæcal valve or first part of the ascending colon.

Attention should again be drawn to the fact that not all the foregoing signs are necessarily characteristic of malignant bowel obstruction; they are most of them true of many forms of serious benign bowel obstruction.

the exploration of the appendicular region. In both instances the colon, distal to the constriction, lost its haustral segmentation and the contents seemed to back up to the "point d'appui"—as if forming a mass, ready to be propelled onwards when the mass movement took place. In both cases a strong mass movement occurred within half an hour, but I did actually see it take place. In one case the whole column on the distal side of the "point d'appui" was swept along, while in the other about half of it was left behind. In the latter case, however, the colon did not look healthy and, in fact, there was mucous colitis. These two cases are, at least, distinctly suggestive that my hypothesis is correct and I am now examining the hepatic flexure both before and after palpation of the cæcum in order to obtain more evidence on the point.

It is too early to make a definite statement but I am becoming more and more convinced that the keystone to the efficiency of this movement of the large intestine lies in the competence of the "point d'appui," and that it is on the competence of this temporary sphincter that the natural action of the bowels depends. Inversely, the failure or incompetency of this valve is responsible for the constipation, *i.e.*, the ineffectiveness of the mass movement when it takes place. For if there is no "point d'appui," the fæces regurgitate into the cæcum, possibly giving rise to the sloppy cæca that we recognise and think of as the *cause* of constipation. Not only so but, in time, one would expect this to lead to inefficiency of the ileo-cæcal valve.

Moreover, if the mass movement is not effective there is nothing except the comparatively feeble movement of the cæcum to propel the food through the large intestine, and when the movements take place they propel the fæces in both directions, *i.e.*, the mass in the cæcum has to act as an inefficient "point d'appui."

CHAPTER XIII.

CONCLUSIONS.*

The progress that has been achieved in the diagnosis of gastric disorders by means of the bismuth method, has been very marked during the five and a half years in which the material for this thesis has been collected. I have been present at as many of the operations as possible, but have had to take my records from the operation books in a very large proportion of cases, and during the first two and a half years the work was anything but encouraging. The opinions I gave as the result of the *x*-ray examination were as often wrong as right, and in many of the failures in diagnosis it seemed that I was hopelessly beside the mark; so much so, that at times I had little confidence in the work and little hope of ever becoming successful, the results were seemingly so contradictory. During the next eighteen months, however, the apparent inconsistencies gradually became fewer in number, but even so it seemed doubtful if the results obtained were worth the time and labour involved, especially when one had to choose between this branch of investigation and others which one knew would yield satisfactory results if one had time to develop them. During the last eighteen months the results have been much more satisfactory, and the operative findings have seldom shown anything that had not been indicated by the *x*-ray examination, although of course I did not always read the signs aright. Some of the failures in diagnosis were in connection with ulceration involving the lesser curvature, which occasionally seems to give rise to no spasmodic contraction; as if some ulcers in this region were either not irritable or interfered with the nerve fibres that should set up localised spasm.

Purely spasmodic contractions have been another cause of failure, but these have very seldom given rise to mistaken diagnosis if the massage test was properly applied, and if the patients were re-examined as a routine procedure, a condition

* Unaltered from first edition.

that is often difficult to hold to in hospital practice owing to the pressure of the waiting list for the surgical beds.

The pars pylorica is still the most difficult region of the stomach about which to obtain reliable information, and the appearances of this part very seldom reveal such details as will assist in the diagnosis. One has therefore to rely almost entirely on the deductive evidence obtained from the rate at which the food leaves the stomach, the tonic action, the peristalsis, secretion, and so forth.

The point has not yet been reached at which it is possible to say, 'there is no active pathological lesion of the stomach walls'—one has so often to make reservations as to the pars pylorica; but, on the other hand, evidence is accumulating that will, I believe, very soon lead to the possibility of always making a definite, positive diagnosis of pyloric ulcers, and when this becomes possible, one will be able to give a definite negative diagnosis in the large number of cases of suspected gastric trouble in which the dyspepsia is functional or secondary to some other trouble.

Defective tonic action is also rather troublesome in making a negative diagnosis, but the appreciation of the changes brought about by defect of tone becomes intuitive after a time, and I am now seldom troubled by the appearances it causes.

The connection between the train of symptoms, which I have discussed under the head of duodenal irritation (p. 99), with other lesions of the intestinal tract is most striking, and confirms the impression that I took from Mr. Moynihan's lecture before the Manchester Pathological Society,* that duodenal ulcer is probably a secondary lesion. As I write I have a patient under observation in whom this train of symptoms is well marked, and the examination I have just made (20 minutes after the food was taken) reveals a large collection of bismuth in the jejunum, and, if confirmed, is almost certainly indicative of adhesions or, as in the last similar case, an ulcer of the jejunum, while the clinical evidence only suggests duodenal ulceration.†

* *Lancet*, February 24, 1912.

† It turned out to be a case of a band of adhesions causing obstruction of the small intestine. There was no evidence of any ulcer to be seen on the duodenal peritoneum.

In taking notes of all the cases the persistent recurrence of a history of severe constipation, especially in women, seems to indicate that the association of gastric lesions with this trouble is more than a coincidence. In many cases also, chiefly men, the teeth have been in a bad state, sometimes actually at the time of the examination, and on several occasions the cleaning up of the mouth has brought about a wonderful change not only in the patient's condition, but also in the x-ray appearances. Cases that have been diagnosed clinically and confirmed radiographically as duodenal ulcer, cases of pyloric obstruction with marked delay in emptying, and spasmodic contractions of the stomach, have all been cured by attention to the teeth and bowels. I have little doubt now that constipation and bad teeth are two of the main factors in determining the onset of ulceration of the stomach and duodenum. Whether or not they are the actual causes I cannot say, but they are both capable of giving rise to spasmodic contractions which have almost all the appearances of pathological changes in the stomach walls. One is more and more impressed with the importance of the physical element of spasm, not only in the causation of true visceral pain, but also in the actual production of some of those lesions of the alimentary canal that were at one time regarded as primary.

One conclusion is quite clear; the more time one spends on a case and the more one considers the x-ray findings in connection with the clinical history, the more accurate will be the diagnosis. The x-ray method is of some value by itself, but when it is taken in conjunction with all the other available means of investigation, it becomes the greatest of all aids we possess in the diagnosis of diseases of the walls of the intestinal tract.

That the work is of value is evidenced by the fact that last year (1911) I examined nearly 350 in-patients by means of the bismuth method at the Royal Infirmary.* But perhaps the most noticeable feature is the fact that one no longer hears of the successful diagnoses but of the failures.

* In 1913 this number had risen to 530, while in the first half of 1914 I had already examined over 350 at the Manchester Royal Infirmary.

There is no useful purpose to be served by collecting statistics and comparing the clinical and x-ray methods of diagnosis in the same series of cases; the requisition cards are made out by house surgeons or house physicians before an exhaustive clinical examination has been made. Moreover, one cannot too strongly urge that the x-ray method is not going to supplant the clinical. It will be the greatest ally that clinical medicine could possibly obtain, and must raise the diagnosis by intelligent guess-work to a surer footing, based on a better understanding of the normal physiology and pathology of the alimentary tract.

NOTE.—There is one fact that has not been noted, and that is the frequency with which one learns that the “patient has been so much better ever since the examination.” Whether this is a psychological effect or due to the large opaque meal I have no means of knowing. So far as my observations go, this effect is most often noted in the cases where ileal stasis has been observed.

CHAPTER XIV.

TABULATION OF CASES.*

To attempt the classification and tabulation of the eight or nine hundred cases of which I have notes was obviously a labour that would yield no definite information. I have therefore only tabulated those in which the actual condition was discovered either on the operating table or in the post-mortem room, and have classed them according to the actual pathological lesion found, and not according to the *x*-ray findings. Even so the task was not easy, for there are many cases in which the operation yields indefinite information as to the exact nature of the lesion. This is especially the case in pyloric obstruction, and for this reason all cases of this nature, whether simple or malignant, are placed under one heading.

Again, description of the operative findings is often so meagre and so lacking in detail that one had to rely in many cases on the memory of the surgeon or house-surgeon for important information. A number of cases have been rejected from this tabulation on the ground of insufficient data as to the operative findings.

Class 1. Cases in which the gastric symptoms were not directly due to a lesion of the stomach or duodenum.

Class 2. Cases in which the symptom-complex of duodenal irritation was found, or in which an actual lesion of the duodenum was discovered.

Class 3. Cases of ulcer of the pars pylorica.

Class 4. Cases of ulceration of the body of the stomach with or without the formation of a definite hour-glass contraction.

* It was obviously impossible, in the circumstances of this publication, to bring this chapter and tabulation up to date. It is therefore left exactly as in the first edition.

Class 5. Cases of carcinoma of the body of the stomach—not including pyloric carcinoma.

Class 6. Cases of pyloric obstruction—whether simple or malignant.

Class 7. Cases in which the stomach was distorted by adhesions—apart from actual disease of the walls.

Class 8. Cases in which surgical interference had not been altogether successful.

I have added introductory remarks to each division with a special note of any case, or class of case, to which I wished to draw attention.

Strictly speaking, classes 1 and 2 should be under one heading, but the symptom-complex of duodenal irritation seems so clear that it will be readily seen why these cases are classed separately.

The clinical diagnosis is taken from the requisition cards which are filled up in the wards.

Tables of Cases

CLASS I. (See p. 57.)

NORMAL.

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
6 53 M.	?	Nil.	No evidence of ulcer.	Gastro-jejunos- tomy did not relieve symp- toms.
25 46 F.	? Carcinoma of stomach	Nil when standing; bismuth segmented over growth when lying down.	Carcinoma of pancreas, etc.; stomach not invaded.	
28 35 F.	? Gastric ulcer; ? carcinoma of stomach.	Slightly atonic; nil else.	No abnormality found.	
54 35 F.	—	Nil but visceroptosis.	Nil in stomach; nephropepy.	Patient no better.
60 33 F.	? Carcinoma; ? gastric ulcer.	Nil except slightly active peristalsis.	Nothing found.	
86 35 M.	Pyloric ulcer.	Excess peristalsis; nil else.	No evidence of abnormality.	
102 22 F.	? Gastric ulcer.	Nil except rather active peristalsis.	Nil except slight adhesions near pylorus; no gastro-jejunos- tomy.	Relieved.
125 39 F.	—	Stomach normal; nil except some air swallow- ing.	Nil found; kidneys fixed.	No benefit.
154 40 F.	?	Stomach appeared to be twisted upon itself; nil else.	Nil found; no gastro-jejunos- tomy.	Patient cured.
171 60 M.	Carcinoma of stomach.	Nil abnormal.	Carcinoma of pancreas.	
182 22 M.	?	Nil abnormal.	Tubercular peritonitis around appendix.	
241 41 M.	Duodenal ulcer.	Nil abnormal.	Surgeon described duodenal scarring, and patient died 3 days later from occult hemor- rhage for which no cause was found.	No duodenal ulceration found at post- mortem.
248 70 M.	Carcinoma.	Nil abnormal found.	Abscess near appendix.	
249 40 F.	Carcinoma of stomach.	Stomach atonic; some visceroptosis.	No abnormalities found, but gastro-jejunos- tomy performed.	
284 25 M.	?	Normal, except rather marked air swallowing.	Appendix removed.	
296 35 F.	Abdom. tumour.	No abnormalities found; no evidence of dis- placements.	No abnormalities found.	Cured.
311 35 M.	? Gastric.	Nil abnormal found.	Appendix thickened with adhesions.	
333 28 F.	Gastric ulcer.	Gastroptosis, but nil else.	Nil in stomach; kidneys low down; nephro- pepy.	

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
337 38 M.	? Gastric ulcer.	Stomach not involved by the growth.	Growth of splenic flexure; adherent to stomach.	
369 44 M.	Dilat. stomach.	Stomach normal, except marked contraction near pylorus; looked like carcinoma.	Nil found; no gastro-jejunostomy.	Spasm cured by laparotomy.
372 24 F.	Dilat. stomach.	Aerophagy; spasm of middle.	Nil found.	
398 38 F.	Dilat. stomach; ? Gastric ulcer.	Hour-glass marked; vomiting from upper sac.	Nothing to account for the hour-glass; appendix removed.	Cured 5 weeks after operation, but relapsed.
402 33 F.	Gastric ulcer.	Nil except active peristalsis.	Appendix removed; nil in stomach.	
405 50 F.	Dilat. stomach.	Nil found.	Nil.	
423 47 M.	Carcinoma? of stomach	Nil, stomach displaced, but not involved.	Growth of colon.	
476 38 M.	? Gastric ulcer.	Nil, peristalsis rather active.	Nil in stomach; chronic appendix.	
478 27 M.	Gastric ulcer.	Nil abnormal.	Nil; old appendix.	
481 64 M.	Carcinoma of stomach.	Nil abnormal noted.	Stone gall-bladder.	
485 30 M.	—	Slightly atonic.	Appendix removed, nil in stomach.	
489 48 M.	Gastric ulcer?	Nil.	Stomach normal; appendix removed.	
497 26 F.	Gastritis.	Irregular card; end looked like adhesions.	Nil.	
555 59 F.	Carcinoma of stomach.	Growth displacing stomach.	Growth of gall-bladder.	
593a47 M.	Pyloric obstruction.	Nil abnormal.	Gall-stones.	
706 57 M.	Duodenal ulcer.	Nil abnormal.	Growth of pancreas.	
612 47 M.	Duodenal ulcer.	Normal.	Few adhesions duodenum to gall-bladder.	
716 27 F.	? Gastric ulcer; ? appendix.	Gastroptosis; nil else.	Stomach normal; inflamed appendix.	
721 36 F.	? Tumour; ?gastric.	Tumour not in relationship to stomach.	Growth of kidneys; stomach not involved.	
761 49 M.	Carcinoma of stomach.	Œsophagus obstructed, with pouching; stomach looks normal.	Stomach normal; gastrostomy.	
772 47 M.	Carcinoma of stomach.	Stomach normal.	Carcinoma of hepatic flexure.	
791 34 F.	? Gastric ulcer.	Stomach normal; rather active secretion; active peristalsis.	Nil found in stomach.	

CLASS II. (See p. 99.)

DUODENAL IRRITATION.

By "normal stomach" in these cases I mean that the organ exhibits either perfect tonic action or is more often hypertonic. The separate bolus in the duodenum refers to the large food shadows seen passing through the duodenum, not to the caput duodeni.

No.	Age	Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
30	30	M.	Dilat. stomach.	Stomach normal, rapid emptying; peristalsis active; separate bolus in duodenum.	Adhesions of duodenum to liver.	
66	31	F.	Gastric ulcer ?	Stomach normal; peristalsis active; separate bolus in duodenum; stomach emptied rather slowly.	Definite cicatrization of duodenum; posterior gastro-jejunostomy.	Cured.
70	37	M.	Duodenal ulcer.	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Cicatrization round duodenum; posterior gastro-jejunostomy.	Cured.
78	28	M.	Tumour.	Stomach normal, rapid emptying; peristalsis active; separate bolus in duodenum.	Extensive carcinoma not involving stomach.	
81	51	M.	Duod. ulcer ?	Stomach normal, rapid emptying; peristalsis active; separate bolus in duodenum, when patient lay down.	Ulcer upper surface duodenum.	Cured.
92	35	M.	Duod. ulcer ?	Stomach normal, rapid emptying; peristalsis active; separate bolus only when patient lay down.	Duodenal ulcer.	Cured.
97	34	F.	? Gastric ulcer.	Stomach normal, rapid emptying; peristalsis active; separate bolus in duodenum; spasm middle stomach.	Cicatrization about duodenum; nil in body of stomach.	Much better, but not cured.
101	62	M.	Duodenal ulcer.	Stomach normal, rapid emptying; peristalsis active; separate bolus in duodenum.	Duodenal ulcer.	Cured.
120	56	M.	Neurosis.	Stomach normal, rapid emptying; peristalsis active; separate bolus in duodenum.	Few adhesions; nil else.	Patient developed G.P. 1.
184	42	M.	Duod. ulcer ?	Stomach normal, rapid emptying; peristalsis active; separate bolus in duodenum.	Duodenal ulcer; gastro-jejunostomy.	Cured.
218	34	M.	Duod. ulcer ?	Stomach normal; peristalsis active; no separate bolus in duodenum; rapid emptying.	Ulceration both sides of pylorus; adhesions to liver.	Relieved, but not cured.
219	22	M.	Gastric ulcer ?	Stomach normal, rapid emptying; peristalsis active; no separate bolus in duodenum.	Appendicular abscess; adhesions.	Patient not cured.

No. Age, Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
240 36 M.	Dilat. stomach.	Visceroptosis; slight atony; peristalsis active.	Duodenal ulcer.	Duodenum not explored.
289 46 M.	Pyloric obstruction.	Stomach normal, rapid emptying; peristalsis active; separate bolus in duodenum.	Growth of lesser curvature	
307 21 F.	Stomach? kidneys?	Stomach normal, except displaced; rapid emptying; peristalsis active; separate bolus in duodenum.	Adhesions to distended gall-bladder.	
314 37 F.	Duodenal ulcer.	Stomach normal, rapid emptying; peristalsis active; separate bolus in duodenum.	Cicatrix round 1st part duodenum.	
401 51 M.	—	Stomach normal, rapid emptying; peristalsis active; separate bolus in duodenum.	Adhesions of duodenum to liver.	
412 54 M.	Gastric ulcer.	Stomach normal; rapid emptying; no active peristalsis; no separate bolus in duodenum.	Duodenal cicatrization.	
417 54 F.	Pyloric obstruction.	Stomach normal; rapid emptying; peristalsis active; no separate bolus in duodenum; small intestine overloaded.	Adhesions of duodenum to liver; small intestine not examined.	
418 50 M.	Carcinoma.	Stomach normal; separate bolus in duodenum; rapid emptying.	Large ulcer lesser curvature, and duodenal ulcer.	No indication of the gastric ulcer.
451 30 M.	Duodenal ulcer.	Stomach normal, rapid emptying; peristalsis active; separate bolus in duodenum.	Adhesion duodenum to gall-bladder; gastro-jejunostomy.	
468 38 M.	Dilat. stomach.	Stomach normal; peristalsis active; no rapid emptying; no separate bolus in duodenum.	Scar on duodenum; gastro-jejunostomy.	
475 37 M.	Pyloric ulcer.	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Duodenal ulcer; gastro-jejunostomy.	
477 35 M.	Gastric ulcer?	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Pericholitis; adhesion to gall-bladder, etc.	
486 34 F.	? Gall-bladder.	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Post-mortem, stone gall-bladder.	
498 22 M.	Gastritis.	Stomach normal; rapid emptying; peristalsis active; separate bolus in duodenum; obstruction at duodeno-jejunal flexure.	Pericholitis; duodenal ulcer; adhesions at duodeno-jejunal flexure.	

No.	Age.	Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
501	30	M.	Gastric ulcer.	Slight at; rapid emptying; peristalsis active; separate bolus in duodenum.	Cicatrical duodenum.	
561	27	M.	Gastric ulcer.	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Duodenal ulcer; gastro-jejunostomy.	
586	40	M.	Duodenal ulcer.	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Thickening of duodenum.	
587	23	M.	Gastritis.	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Duodenal ulcer; gastro-jejunostomy.	
588	35	M.	Gastric ulcer?	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Adhesions duodenum to gall-bladder and colon.	
589	31	M.	Duodenal ulcer.	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Duodenal ulcer.	
613	50	M.	Duodenal ulcer.	Stomach normal; peristalsis active; rapid emptying; bolus in duodenum, which does not move on.	Cicatrices of pylorus and duodenum.	
619	36	M.	Gall-stones.	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Gall-stones; adhesions.	
638	29	M.	Dyspepsia.	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Long appendix fixed by adhesions very high up.	
658	24	M.	? Duod. ulcer.	Stomach normal; peristalsis active; rapid emptying; coils of jejunum seen in left iliac fossa.	Duodenal ulcer and ring ulcer of jejunum (tubercular).	Duodenal ulcer and ring ulcer of jejunum.
730	49	F.	Dilat. stomach.	Gastropotosis; nil else.	Nil in stomach; adhesions to duodenum.	
749	56	F.	Dyspepsia.	Stomach normal; peristalsis active; rapid emptying; no separate bolus in duodenum.	Gall-stones and adhesions.	

CLASS III. (See p. 70.)

ULCER OF PYLORIC REGION.

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
143 55 F.	? Pyloric obstruction; dilated stomach.	Large atonic stomach; retained food; no delay in passing food out.	Cicatrization about pylorus; small active ulcer.	Cured.
308 42 M.	Pyloric ulcer.	Large atonic stomach with retained fluid; no marked delay emptying.	Cicatrix near pylorus.	
335 39 F.	? Gastric ulcer.	Hour-glass; secretion into upper sac; delay in emptying lower sac (24 hours).	Typical hour-glass stomach; ulceration and cicatrization of pylorus.	Secretion into upper sac.
396 44 M.	Dilat. stomach.	Atony; hypersecretion.	Thickening of pylorus; gastro-jejunostomy.	
579 40 F.	Gastric ulcer.	Hour-glass; cicatrization; hypersecretion to upper sac.	'As though string had been tied round stomach'; also pyloric ulcer.	Hypersecretion with pyloric ulcer.
593 31 M.	Duodenal ulcer.	Stomach normal; hypersecretion; peristalsis active; no food seen passing through duodenum; ? delay emptying.	Thickening about pylorus and lesser curvature.	Hypersecretion with ulcer of pylorus.
666 32 M.	Carcinoma.	Slight atony; nil else noted.	Ulcer posterior wall 1 inch from pylorus.	
717 36 F.	Gastric ulcer.	Hour-glass; excessive secretion upper sac.	Cicatrical hour-glass; ulcer of pylorus.	
720 40 F.	Gastritis.	Well-marked hour-glass; pyloric obstruction; rapid secretion.	Mass of adhesions, middle lesser curvature; thickened pylorus with active ulcer; inflamed appendix.	Rapid secretion with ulcer of pylorus.
723 32 M.	Duodenum? appendix?	Hour-glass (spasmodic); pain relieved when food passed through; active secretion; active peristalsis.	No hour-glass; no ulcer; many adhesions about pylorus and ? ulcer pylorus; adhesions appendix.	
724 24 M.	? Gastric ulcer; ? carcinoma pylorus.	Normal; active secretion; peristalsis active.	Cicatrix about pylorus; active ulcer.	
777 32 M.	Dilat. stomach.	Stomach normal; excessive secretion; active peristalsis.	Ulcer pyloric portion on posterior wall.	

CLASS IV. (See p. 78.) ULCERATION OF THE BODY OF THE STOMACH.

HOURL-GLOSS CONTRACTIONS.

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
24 50 M.	? Gastric ulcer.	Slight delay in emptying; nil else.	Large ulcer middle lesser curvature; thickening about pylorus.	Patient cured by gastro-jejunostomy.
35 32 F.	? Pyloric obstruction.	Definite hour-glass and pyloric obstruction.	Typical hour-glass; contraction and thickening of pylorus.	Cured by gastro-troplasty and gastro-jejunostomy.
37 34 M.	—	Hour-glass, partly spasmodic.	Ulcer lesser curvature; some cicatrization.	Excised.
79 52 M.	Gastric ulcer.	Spasmodic hour-glass, which relaxed leaving indentation.	Ulcer anterior wall; excised.	
83 48 F.	Obstruction of pylorus.	Definite hour-glass small channel; definite delay in lower sac.	Cicatricial hour-glass; cicatricial pylorus; gastro-troplasty and gastro-jejunostomy.	Cured.
89 47 M.	Growth?	Atonic with spasm about middle.	Large stomach; cicatrix on lesser curvature; gastro-jejunostomy.	Cured.
91 42 F.	Pyloric obstruction?	Cicatricial hour-glass (funnel-shaped); no pyloric obstruction.	Ulcer greater curvature; cicatricial hour-glass, with adhesion.	Cured.
93 41 M.	Œsophageal obstruction	Huge pouching just above diaphragm.	Post-mortem, small ulcer anterior surface near cardiac orifice.	No dilatation of œsophagus and no obstruction found post-mortem.
137 42 F.	Dilatation.	Hour-glass stomach; upper sac only seen.	Cicatricial hour-glass.	Patient died suddenly three weeks later.
193 59 F.	Gastric ulcer.	Atonic; slight delay emptying; some obstruction duodenum.	Ulcer 1 inch below cardiac orifice; ulcer just beyond pylorus with cicatricial contraction.	
203 63 F.	—	Hour-glass, and distorted by adhesions; lower sac in right iliac fossa; delay in emptying of lower sac.	Ulcers and cicatrices middle of stomach; growth at pylorus.	
207 30 F.	Gastric ulcer; gastro-jejunostomy 1908.	Well-marked cicatricial hour-glass; stomach working perfectly.	Stomach not explored, but kidney fixed November, 1909.	Unrelieved; entered also in Class VIII.

No.	Age.	Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
207	31	F.	Same case one year later.	Exactly same picture as above.	Stomach almost divided by marked contraction.	Cured.
230	39	F.	? Stenosis of pylorus.	Hour-glass stomach, and definite delay in emptying.	Ring of ulceration of greater curvature; constriction of pylorus.	
239	40	F.	Gastric ulcer.	Funnel-shaped hour-glass; inverted peristalsis in lower sac; delay in emptying (24 hours).	Multiple scars forming trilobular stomach; growth at pylorus.	
266	43	M.	Old gastro-jejunostomy.	Hour-glass stomach well marked; stoma working perfectly.	Hour-glass, with active ulcer.	Cured by gastropasty; a small ulcer of body had been noted at time of first operation.
286	39	F.	Dilat. stomach? gastric ulcer.	Hour-glass stomach; no pyloric obstruction.	Hour-glass; probably active ulcer.	
299	23	F.	Gastric ulcer.	Hour-glass stomach, partly spasmodic; no pyloric obstruction.	Large ulcer lesser curvature; some puckering; gastro-jejunostomy to upper pouch.	
310	40	F.	Gastric ulcer; ? pyloric obstruction.	Hour-glass stomach; definite delay emptying.	Trilobular stomach; cicatricial pyloric obstruction.	
331	29	F.	Gastric ulcer.	Division of stomach near pylorus.	Hour-glass stomach.	
335	39	F.	? Gastric ulcer; ? carcinoma.	Hour-glass; secretion into upper sac; delay in emptying lower sac (24 hours).	Typical hour-glass stomach; ulceration and cicatrization of pylorus.	Lower sac quite large. Secretion into upper sac; also entered in Class III.
364	37	F.	Pyloric stenosis.	Spasmodic hour-glass; no pyloric obstruction.	Cicatricial hour-glass, with active ulcer.	
367	43	F.	Dilat. stomach.	Hour-glass, chiefly organic; no pyloric obstruction.	Large; gastro-jejunostomy lesser curvature, with cicatrization.	
371	39	F.	Gastric ulcer.	Hour-glass; no pyloric obstruction.	Hour-glass; gastro-jejunostomy to lower sac.	
389	40	F.	Gastric ulcer; old gastro-jejunostomy.	Hour-glass marked; stoma patent.	Hour-glass, with ulcer; gastropasty.	
400	33	F.	Gall-stone? kidney; old gastro-jejunostomy.	Perfect hour-glass; stoma patent.	Cicatricial hour-glass.	
406	26	F.	Old gastro-jejunostomy	Hour-glass, chiefly spasmodic; stoma patent.	Hour-glass, cicatricial, above stoma.	
418	50	M.	Carcinoma.	Nil; separate bolus in duodenum.	Large ulcer lesser curvature; cicatrix in duodenum.	
420	53	F.	? Stomach; ? Kidneys.	Spasmodic hour-glass, and separate bolus in duodenum.	Hour-glass and duodenal ulcer.	
455	48	F.	—	Cicatricial hour-glass.	Cicatricial hour-glass.	

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
461 39 F.	Gastric ulcer.	Marked hour-glass.	Cicatrical hour-glass.	
465 39 F.	Gastric ulcer.	Incomplete hour-glass; delayed emptying lower sac.	Pyloric obstruction; old cicatrices anterior walls of stomach.	
482 29 F.	Gastric ulcer; pyloric obstruction.	Hour-glass (cicatrical); no pyloric obstruction.	Cicatrical hour-glass. Thickening of pylorus.	
490 24 F.	Dilat. stomach.	Spasmodic hour-glass; delayed emptying lower sac.	Cicatix of greater curvature and also at pylorus.	
492 31 F.	Neurosis.	Hour-glass, and delayed emptying lower sac.	Hour-glass, adherent to liver; pyloric obstruction.	
493 45 F.	Abdominal tumour.	Hour-glass; no evidence of growth.	Typical hour-glass; no pyloric obstruction or growth.	
573 18 F.	Gastric ulcer.	Hour-glass; some pyloric obstruction.	Ulcer lesser curvature; thickened pylorus.	
579 40 F.	? Gastric ulcer.	Hour-glass, cicatrical; hypersecretion to upper sac.	'As though string had been tied round stomach'; also pyloric ulcer.	Hypersecretion with pyloric ulcer.
583 28 F.	Gastric ulcer.	Spasmodic hour-glass.	Large ulcer middle stomach.	
589 39 F.	Pyloric stenosis.	Hour-glass, cicatrical.	Cicatrical hour-glass.	
618 36 M.	Gastric ulcer.	Hour-glass, chiefly spasmodic.	Ulcer greater curvature.	
686 34 F.	Gastric ulcer?	Hour-glass, partly spasmodic; no pyloric obstruction.	Large ulcer lesser curvature; cicatrization.	
695 45 F.	Dilat. stomach; gastric ulcer?	Hour-glass, cicatrical, penetrating ulcer; retained food (24 hours).	Mass of adhesions middle stomach; hour-glass; pylorus normal; anterior gastrojejunostomy.	
717 36 F.	Gastric ulcer.	Hour-glass; excessive secretion upper sac.	Cicatrical hour-glass; ulcer of pylorus.	
720 40 F.	Gastritis.	Well-marked hour-glass; pyloric obstruction; rapid secretion.	Mass of adhesions middle lesser curvature; thickened pylorus with active ulcer; inflamed appendix.	No pyloric obstruction, although retained food.
760 42 F.	Gastric? kidney.	Gastroptosis; pylorus dropped 2 inches; nephroptosis.	Cicatrical ulcer lesser curvature, 2 inches from pylorus.	Hypersecretion with ulcer of pylorus.
782 65 F.	Gastric ulcer.	Pyloric portion sacculated and bound to liver; upper portion suggested hour-glass contraction.	Mass of adhesions of pyloric portion stomach to liver; old ulceration; ulcer of pylorus.	Rapid secretion with ulcer of pylorus.

CLASS V. (See p. 85.)

CARCINOMA OF THE STOMACH.

No.	Age.	Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
7	43	M.	Carcinoma of stomach?	Very excessive peristalsis; pars pylorica obliterated. No delay in emptying.	Annular growth of pylorus.	Excised growth; patient died 18 months later.
8	35	M.	Carcinoma of stomach.	Stomach invaded; small irregular cavity only left.	Advanced carcinoma; inoperable.	
9	43	M.	Carcinoma of stomach.	Inroads of growth well defined.	Inoperable carcinoma.	
21	45	M.	—	Irregularity in outline of greater curvature.	Inoperable carcinoma.	
40	37	M.	? Carcinoma.	Irregularities of outline.	Advanced carcinoma.	
42	53	F.	? Carcinoma of stomach.	Inroads of growth near pylorus; retention of food.	Carcinoma of pylorus; gastro-jejunostomy.	
48	52	F.	Carcinoma of stomach.	Esophageal obstruction at cardiac orifice; no evidence of growth of stomach.	Post-mortem six weeks later; growth of lesser curvature 5" X 6"; cardiac orifice not involved.	No contraction of cardiac orifice post-mortem.
58	54	M.	? Carcinoma of stomach; ? aneurism.	Light areas in midst of bismuth shadow.	Inoperable carcinoma; posterior wall chiefly.	Patient died 3 months later.
64	39	M.	? Carcinoma of stomach.	Irregular in outline.	Advanced carcinoma.	
170	58	M.	? Carcinoma of stomach.	Inroads of growth giving hour-glass appearance.	Inoperable carcinoma.	
233	53	F.	? Carcinoma of stomach.	Definite irregularities of outline.	Mass of growth in lesser curvature.	
289	46	M.	? Pyloric obstruction.	Stomach normal; shadows well seen in duodenum; active peristalsis.	Plaque of growth on lesser curvature; duodenum not explored.	No evidence of growth of stomach.

No.	Age.	Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
297	34	M.	Pyloric ulcer.	Irregular in outline.	Growth lesser curvature.	
300	29	F.	? Carcinoma of stomach.	Large atonic stomach; nil else.	Operation six months later; large mass involving anterior wall.	
342	66	F.	Carcinoma of stomach.	Gastric cavity obliterated, except along greater curvature.	Post-mortem; massive carcinoma involving whole stomach.	
366	55	F.	Abdom. tumour.	Obliteration of all the cavity, except greater curvature.	Large mass extending from lesser curvature.	
380	43	M.	Duodenal ulcer.	-Stomach cavity small and irregular; back pressure oesophageal dilatation.	Stomach one mass of growth.	
385	55	F.	Carcinoma of stomach.	Nil abnormal made out.	Growth lesser curvature, size of Tangerine.	
403	50	M.	—	Irregularity of pyloric portion.	Post-mortem; carcinoma.	
469	66	M.	? Carcinoma.	Cavity nearly obliterated.	Large growth; inoperable.	
582	52	F.	Carcinoma of ascending colon.	Cavity nearly obliterated; right side diaphragm pushed up.	Growth involving colon and stomach, secondary in liver.	
614	60	M.	? Carcinoma of pylorus.	Irregularities near pylorus; no obstruction.	Carcinoma of pylorus.	
776	47	M.	? Gastric ulcer; ? pyloric stenosis.	Marked inroad of pyloric portion; retained food.	Carcinoma of lesser curvature and pylorus.	Patient died 36 hours later, cause unknown.

CLASS VI. (See p. 69.) PYLORIC OBSTRUCTION.

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
10 35 F.	Dyspepsia.	Complete atony; bismuth seen in stomach for 5 days after examination.	Pyloric obstruction (cicatrical); gastro-jejuno-ostomy.	Not cured.
20 52 M.	? Carcinoma.	Normal stomach, full of fluid; bismuth in stomach after 24 hours.	Carcinoma of pylorus with ulceration.	
24 50 M.	Gastric ulcer.	Slight delay in emptying.	Ulcer of lesser curvature and thickening of pylorus; gastro-jejunostomy.	Cured. See also under Class IV.
29 54 M.	? Carcinoma of stomach.	Marked atony; retained fluid; slight delay.	Ulcer surrounding pylorus; gastro-jejunostomy.	
31 55 F.	? Carcinoma of stomach.	Retained food; tone perfect; bismuth retained 24 hours.	Extensive carcinoma involving pylorus; gastro-jejunostomy.	Patient cured.
41 32 F.	—	Hour-glass, partly spasmodic; peristalsis of upper sac; delayed emptying of lower sac.	Pyloric thickening; no ulcer of body.	Not cured; patient persisted in vomiting from upper sac.
56 44 F.	? Gastric ulcer.	Visceroptosis; delay in emptying; marked peristalsis.	Thickening of pylorus; gastro-jejunostomy.	Cured.
65 25 F.	? Gastric ulcer.	Some delay in emptying, in spite of excessive peristalsis; some visceroptosis.	Thickening about pylorus; gastro-jejunostomy.	Relieved by operation.
72 61 F.	Gastric ulcer.	Extreme atony; marked peristalsis; delayed emptying (24 hours).	Thickening about pylorus; gastro-jejunostomy.	Cured.
80 46 M.	? Pyloric obstruction.	Retained food; perfect tone; defective peristalsis; delayed emptying (24 hours).	Large mass about pylorus.	
88 47 F.	Carcinoma of pylorus.	Stomach atonic; marked delay emptying (24 hours).	Growth at pylorus; huge stomach; gastro-jejunostomy.	'Cured' 2 years later.
94 32 M.	Dilat. stomach.	Definite delay in emptying (12 hours); some atony.	Thickening of pylorus.	Cured.
98 43 F.	? Gastric ulcer.	Large atonic stomach with retained food; slight delay in emptying; marked peristalsis.	Adhesions along lesser curvature and pylorus; ? as to obstruction.	Cured.
126 46 M.	Duodenal ulcer.	Nil, except rather active peristalsis; stomach emptying rapidly.	9 months later pyloric obstruction found.	Rapid emptying noted in early stage.
175 42 M.	Gastric ulcer.	Definite delay in emptying; some atony.	Ulcer of pylorus; adhesions to liver.	Cured.
179 34 F.	—	Very atonic stomach; active peristalsis; slight delay emptying.	Pyloric thickening and ulcer; stomach very large.	Cured.

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
192 51 M.	—	Retained food; delay in emptying.	Growth about pylorus and liver.	
201 48 M.	? Duod. ulcer; ? malignant.	Definite delay (24 hours); stomach normal; retained food; peristalsis not active.	Cicatrix of pylorus; gastro-jejunostomy.	Cured.
205 36 M.	Duodenal ulcer.	Stomach normal; violent peristalsis occasionally; slight delayed emptying (10 hours).	Old cicatrices about pylorus and duodenum; gastro-jejunostomy.	Patient died 3 days after operation; no cause known.
208 35 M.	Pyloric obstruction.	Retained food 24 hours; perfect tone; peristalsis absent.	Ring of ulceration round pylorus.	Cured.
217 36 F.	Carcinoma of stomach.	Huge atonic stomach; definite delay in emptying (24 hours).	Huge stomach; growth around pylorus.	
220 49 M.	Duodenal ulcer.	Stomach normal; very active peristalsis; occasionally some delay emptying.	Adhesion of pylorus and duodenum to gall-bladder; ? pyloric obstruction.	
226 29 M.	Duodenal ulcer.	Stomach toneless; definite delay (24 hours).	Thickening of pylorus and scarring of duodenum.	Cured.
230 39 F.	? Pyloric stenosis.	Hour-glass stomach and definite delay in emptying lower sac.	Ring of ulceration greater curvature and constriction of pylorus.	
239 40 F.	Gastric ulcer.	Funnel-shaped hour-glass; inverted peristalsis in lower sac; delay in emptying (24 hours).	Multiple scars forming trilobular stomach; growth at pylorus.	
250 49 M.	Dilat. stomach.	Huge distended stomach full of food; marked delay (48 hours).	Large growth of pylorus.	
269 40 F.	? Dilated stomach; pyloric stenosis.	Complete atony; delay in emptying (24 hours); incomplete hour-glass.	Cicatrices about pylorus and smaller one on greater curvature.	Cured.
272 43 M.	Duodenal ulcer.	Stomach, normal, but occasionally active peristalsis.	Adhesions and glands malignant? about pylorus.	
293 35 F.	Dilat. stomach.	Extreme atony; delay emptying.	Growth at pylorus; gastro-jejunostomy.	Patient cured (6 months).
294 43 M.	Gastric ulcer.	Slight atony; occasional violent waves of peristalsis; no marked delay emptying.	Thickening pylorus; gastro-jejunostomy.	Cured.
295 50 M.	? Carcinoma of stomach.	Definite delay in emptying; occasional violent peristalsis; inroads of growth at pylorus.	Growth about pylorus.	
309 56 M.	? hour-glass.	Retained fluid; perfect tone; peristalsis absent; definite delay (24 hours).	Large growth around pylorus; gastro-jejunostomy.	

No. Age, Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
240 36 M.	Dilat. stomach.	Visceroptosis; slight atony; peristalsis active.	Duodenal ulcer.	
289 46 M.	Pyloric obstruction.	Stomach normal, rapid emptying; peristalsis active; separate bolus in duodenum.	Growth of lesser curvature	Duodenum not explored.
307 31 F.	Stomach? kidneys?	Stomach normal, except displaced; rapid emptying; peristalsis active; separate bolus in duodenum.	Adhesions to distended gall-bladder.	
314 37 F.	Duodenal ulcer.	Stomach normal, rapid emptying; peristalsis active; separate bolus in duodenum.	Cicatrix round 1st part duodenum.	
401 51 M.	—	Stomach normal, rapid emptying; peristalsis active; separate bolus in duodenum.	Adhesions of duodenum to liver.	
412 54 M.	Gastric ulcer.	Stomach normal; rapid emptying; no active peristalsis; no separate bolus in duodenum.	Duodenal cicatrization.	
417 54 F.	Pyloric obstruction.	Stomach normal; rapid emptying; peristalsis active; no separate bolus in duodenum; small intestine overloaded.	Adhesions of duodenum to liver; small intestine not examined.	
418 50 M.	Carcinoma.	Stomach normal; separate bolus in duodenum; rapid emptying.	Large ulcer lesser curvature, and duodenal ulcer.	No indication of the gastric ulcer.
451 30 M.	Duodenal ulcer.	Stomach normal, rapid emptying; peristalsis active; separate bolus in duodenum.	Adhesion duodenum to gall-bladder; gastro-jejunostomy.	
468 38 M.	Dilat. stomach.	Stomach normal; peristalsis active; no rapid emptying; no separate bolus in duodenum.	Scar on duodenum; gastro-jejunostomy.	
475 37 M.	Pyloric ulcer.	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Duodenal ulcer; gastro-jejunostomy.	
477 35 M.	Gastric ulcer?	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Pericholitis; adhesion to gall-bladder, etc.	
486 34 F.	? Gall-bladder.	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Post-mortem, stone gall-bladder.	
498 22 M.	Gastritis.	Stomach normal; rapid emptying; peristalsis active; separate bolus in duodenum; obstruction at duodeno-jejunal flexure.	Pericholitis; duodenal ulcer; adhesions at duodeno-jejunal flexure.	

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
501 30 M.	Gastric ulcer.	Slight at; rapid emptying; peristalsis active; separate bolus in duodenum.	Cicatricial duodenum.	
561 27 M.	Gastric ulcer.	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Duodenal ulcer; gastro-jejunostomy.	
586 40 M.	Duodenal ulcer	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Thickening of duodenum.	
587 23 M.	Gastritis.	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Duodenal ulcer; gastro-jejunostomy.	
588 35 M.	Gastric ulcer?	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Adhesions duodenum to gall-bladder and colon.	
589 31 M.	Duodenal ulcer.	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Duodenal ulcer.	
613 50 M.	Duodenal ulcer.	Stomach normal; peristalsis active; rapid emptying; bolus in duodenum, which does not move on.	Cicatrices of pylorus and duodenum.	
619 36 M.	Gall-stones.	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Gall-stones; adhesions.	
638 29 M.	Dyspepsia.	Stomach normal; peristalsis active; rapid emptying; separate bolus in duodenum.	Long appendix fixed by adhesions very high up.	
658 24 M.	? Duod. ulcer.	Stomach normal; peristalsis active; rapid emptying; coils of jejunum seen in left iliac fossa.	Duodenal ulcer and ring ulcer of jejunum (tubercular).	Duodenal ulcer and ring ulcer of jejunum.
730 49 F.	Dilat. stomach.	Gastroptosis; nil else.	Nil in stomach; adhesions to duodenum.	
749 56 F.	Dyspepsia.	Stomach normal; peristalsis active; rapid emptying; no separate bolus in duodenum.	Gall-stones and adhesions.	

CLASS III. (See p. 70.)

ULCER OF PYLORIC REGION.

No.	Age.	Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
143	55	F.	? Pyloric obstruction; dilated stomach.	Large atonic stomach; retained food; no delay in passing food out.	Cicatrizization about pylorus; small active ulcer.	Cured.
308	42	M.	Pyloric ulcer.	Large atonic stomach with retained fluid; no marked delay emptying.	Cicatrix near pylorus.	
335	39	F.	? Gastric ulcer.	Hour-glass; secretion into upper sac; delay in emptying lower sac (24 hours).	Typical hour-glass stomach; ulceration and cicatrization of pylorus.	Secretion into upper sac.
396	44	M.	Dilat. stomach.	Atony; hypersecretion.	Thickening of pylorus; gastro-jejunostomy.	
579	40	F.	Gastric ulcer.	Hour-glass; cicatrization; hypersecretion to upper sac.	'As though string had been tied round stomach'; also pyloric ulcer.	Hypersecretion with pyloric ulcer.
593	31	M.	Duodenal ulcer.	Stomach normal; hypersecretion; peristalsis active; no food seen passing through duodenum; ? delay emptying.	Thickening about pylorus and lesser curvature.	Hypersecretion with ulcer of pylorus.
666	32	M.	Carcinoma.	Slight atony; nil else noted.	Ulcer posterior wall 1 inch from pylorus.	
717	36	F.	Gastric ulcer.	Hour-glass; excessive secretion upper sac.	Cicatricial hour-glass; ulcer of pylorus.	
720	40	F.	Gastritis.	Well-marked hour-glass; pyloric obstruction; rapid secretion.	Mass of adhesions, middle lesser curvature; thickened pylorus with active ulcer; inflamed appendix.	Rapid secretion with ulcer of pylorus.
723	32	M.	Duodenum? appendix?	Hour-glass (spasmodic); pain relieved when food passed through; active secretion; active peristalsis.	No hour-glass; no ulcer; many adhesions about pylorus and ? ulcer pylorus; adhesions appendix.	
724	24	M.	? Gastric ulcer; ? carcinoma pylorus.	Normal; active secretion; peristalsis active.	Cicatrix about pylorus; active ulcer.	
777	32	M.	Dilat. stomach.	Stomach normal; excessive secretion; active peristalsis.	Ulcer pyloric portion on posterior wall.	

CLASS IV. (See p. 78.) ULCERATION OF THE BODY OF THE STOMACH.

HOURL-GLASS CONTRACTIONS.

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
24 50 M.	? Gastric ulcer.	Slight delay in emptying; nil else.	Large ulcer middle lesser curvature; thickening about pylorus.	Patient cured by gastro-jejunostomy.
35 32 F.	? Pyloric obstruction.	Definite hour-glass and pyloric obstruction.	Typical hour-glass; contraction and thickening of pylorus.	Cured by gastrotomy and gastro-jejunostomy.
37 34 M.	—	Hour-glass, partly spasmodic.	Ulcer lesser curvature; some cicatrization.	Excised.
79 52 M.	Gastric ulcer.	Spasmodic hour-glass, which relaxed leaving indentation.	Ulcer anterior wall; excised.	
83 48 F.	Obstruction of pylorus.	Definite hour-glass small channel; definite delay in lower sac.	Cicatrical hour-glass; cicatrical pylorus; gastrotomy and gastro-jejunostomy.	Cured.
89 47 M.	Growth?	Atonic with spasm about middle.	Large stomach; cicatrix on lesser curvature; gastro-jejunostomy.	Cured.
91 42 F.	Pyloric obstruction?	Cicatrical hour-glass (funnel-shaped); no pyloric obstruction.	Ulcer greater curvature; cicatrical hour-glass, with adhesion.	Cured.
93 41 M.	Esophageal obstruction	Huge pouching just above diaphragm.	Post-mortem, small ulcer anterior surface near cardiac orifice.	No dilatation of cesophagus and no obstruction found post-mortem.
137 42 F.	Dilatation.	Hour-glass stomach; upper sac only seen.	Cicatrical hour-glass.	Patient died suddenly three weeks later.
193 59 F.	Gastric ulcer.	Atonic; slight delay emptying; some obstruction duodenum.	Ulcer 1 inch below cardiac orifice; ulcer just beyond pylorus with cicatrical contraction.	
203 63 F.	—	Hour-glass, and distorted by adhesions; lower sac in right iliac fossa; delay in emptying of lower sac.	Ulcers and cicatrices middle of stomach; growth at pylorus.	
207 30 F.	Gastric ulcer; gastro-jejunostomy 1908.	Well-marked cicatrical hour-glass; stomach working perfectly.	Stomach not explored, but kidney fixed November, 1909.	Unrelieved; entered also in Class VIII.

No.	Age.	Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
207	31	F.	Same case one year later.	Exactly same picture as above.	Stomach almost divided by marked contraction.	Cured.
230	39	F.	? Stenosis of pylorus.	Hour-glass stomach, and definite delay in emptying.	Ring of ulceration of greater curvature; constriction of pylorus.	
239	40	F.	Gastric ulcer.	Funnel-shaped hour-glass; inverted peristalsis in lower sac; delay in emptying (24 hours).	Multiple scars forming trilobular stomach; growth at pylorus.	
266	43	M.	Old gastro-jejunostomy.	Hour-glass stomach well marked; stoma working perfectly.	Hour-glass, with active ulcer.	Cured by gastrotomy; a small ulcer of body had been noted at time of first operation.
286	39	F.	Dilat. stomach? gastric ulcer.	Hour-glass stomach; no pyloric obstruction.	Hour-glass; probably active ulcer.	
299	23	F.	Gastric ulcer.	Hour-glass stomach, partly spasmodic; no pyloric obstruction.	Large ulcer lesser curvature; some puckering; gastro-jejunostomy to upper pouch.	
310	40	F.	Gastric ulcer; ? pyloric obstruction.	Hour-glass stomach; definite delay emptying.	Trilobular stomach; cicatricial pyloric obstruction.	
331	29	F.	Gastric ulcer.	Division of stomach near pylorus.	Hour-glass stomach.	
335	39	F.	? Gastric ulcer; ? carcinoma.	Hour-glass; secretion into upper sac; delay in emptying lower sac (24 hours).	Typical hour-glass stomach; ulceration and cicatrization of pylorus.	Lower sac quite large. Secretion into upper sac; also entered in Class III.
364	37	F.	Pyloric stenosis.	Spasmodic hour-glass; no pyloric obstruction.	Cicatricial hour-glass, with active ulcer.	
367	43	F.	Dilat. stomach.	Hour-glass, chiefly organic; no pyloric obstruction.	Large; gastro-jejunostomy lesser curvature, with cicatrization.	
371	39	F.	Gastric ulcer.	Hour-glass; no pyloric obstruction.	Hour-glass; gastro-jejunostomy to lower sac.	
389	40	F.	Gastric ulcer; old gastro-jejunostomy.	Hour-glass marked; stoma patent.	Hour-glass, with ulcer; gastrotomy.	
400	33	F.	Gall-stone? kidney; old gastro-jejunostomy.	Perfect hour-glass; stoma patent.	Cicatricial hour-glass.	
406	26	F.	Old gastro-jejunostomy	Hour-glass, chiefly spasmodic; stoma patent.	Hour-glass, cicatricial, above stoma.	
418	50	M.	Carcinoma.	Nil; separate bolus in duodenum.	Large ulcer lesser curvature; cicatrix in duodenum.	
420	53	F.	? Stomach; ? Kidneys.	Spasmodic hour-glass, and separate bolus in duodenum.	Hour-glass and duodenal ulcer.	
455	48	F.	—	Cicatricial hour-glass.	Cicatricial hour-glass.	

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
461 39 F.	Gastric ulcer.	Marked hour-glass.	Cicatrical hour-glass.	
465 39 F.	Gastric ulcer.	Incomplete hour-glass; delayed emptying lower sac.	Pyloric obstruction; old cicatrices anterior walls of stomach.	
482 29 F.	Gastric ulcer; pyloric obstruction.	Hour-glass (cicatrical); no pyloric obstruction.	Cicatrical hour-glass. Thickening of pylorus.	
490 24 F.	Dilat. stomach.	Spasmodic hour-glass; delayed emptying lower sac.	Cicatix of greater curvature and also at pylorus.	
492 31 F.	Neurosis.	Hour-glass, and delayed emptying lower sac.	Hour-glass, adherent to liver; pyloric obstruction.	
493 45 F.	Abdominal tumour.	Hour-glass; no evidence of growth.	Typical hour-glass; no pyloric obstruction or growth.	
573 18 F.	Gastric ulcer.	Hour-glass; some pyloric obstruction.	Ulcer lesser curvature; thickened pylorus.	
579 40 F.	? Gastric ulcer.	Hour-glass, cicatrical; hypersecretion to upper sac.	'As though string had been tied round stomach'; also pyloric ulcer.	Hypersecretion with pyloric ulcer.
583 28 F.	Gastric ulcer.	Spasmodic hour-glass.	Large ulcer middle stomach.	
589 39 F.	Pyloric stenosis.	Hour-glass, cicatrical.	Cicatrical hour-glass.	
618 36 M.	Gastric ulcer.	Hour-glass, chiefly spasmodic.	Ulcer greater curvature.	
686 34 F.	Gastric ulcer?	Hour-glass, partly spasmodic; no pyloric obstruction.	Large ulcer lesser curvature; cicatrization.	
695 45 F.	Dilat. stomach; gastric ulcer?	Hour-glass, cicatrical, penetrating ulcer; retained food (24 hours).	Mass of adhesions middle stomach; hour-glass; pylorus normal; anterior gastro-jejunostomy.	
717 36 F.	Gastric ulcer.	Hour-glass; excessive secretion upper sac.	Cicatrical hour-glass; ulcer of pylorus.	
720 40 F.	Gastritis.	Well-marked hour-glass; pyloric obstruction; rapid secretion.	Mass of adhesions middle lesser curvature; thickened pylorus with active ulcer; inflamed appendix.	No pyloric obstruction, although retained food.
760 42 F.	Gastric? kidney.	Gastroptosis; pylorus dropped 2 inches; nephroptosis.	Cicatrical ulcer lesser curvature, 2 inches from pylorus.	Hypersecretion with ulcer of pylorus.
782 65 F.	Gastric ulcer.	Pyloric portion sacculated and bound to liver; upper portion suggested hour-glass contraction.	Mass of adhesions of pyloric portion stomach to liver; old ulceration; ulcer of pylorus.	Rapid secretion with ulcer of pylorus.

CLASS V. (See p. 85.) CARCINOMA OF THE STOMACH.

No.	Age.	Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
7	43	M.	Carcinoma of stomach?	Very excessive peristalsis; pars pylorica obliterated. No delay in emptying.	Annular growth of pylorus.	Excised growth; patient died 18 months later.
8	35	M.	Carcinoma of stomach.	Stomach invaded; small irregular cavity only left.	Advanced carcinoma; inoperable.	
9	43	M.	Carcinoma of stomach.	Inroads of growth well defined.	Inoperable carcinoma.	
21	45	M.	—	Irregularity in outline of greater curvature.	Inoperable carcinoma.	
40	37	M.	? Carcinoma.	Irregularities of outline.	Advanced carcinoma.	
42	53	F.	? Carcinoma of stomach.	Inroads of growth near pylorus; retention of food.	Carcinoma of pylorus; gastro-jejunostomy.	
48	52	F.	Carcinoma of stomach.	Esophageal obstruction at cardiac orifice; no evidence of growth of stomach.	Post-mortem six weeks later; growth of lesser curvature 5" X 6"; cardiac orifice not involved.	No contraction of cardiac orifice post-mortem.
58	54	M.	? Carcinoma of stomach; ? aneurism.	Light areas in midst of bismuth shadow.	Inoperable carcinoma; posterior wall chiefly.	Patient died 3 months later.
64	39	M.	? Carcinoma of stomach.	Irregular in outline.	Advanced carcinoma.	
170	58	M.	? Carcinoma of stomach.	Inroads of growth giving hour-glass appearance.	Inoperable carcinoma.	
233	53	F.	? Carcinoma of stomach.	Definite irregularities of outline.	Mass of growth in lesser curvature.	
239	46	M.	? Pyloric obstruction.	Stomach normal; shadows well seen in duodenum; active peristalsis.	Plaque of growth on lesser curvature; duodenum not explored.	No evidence of growth of stomach.

No.	Age.	Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
297	34	M.	Pyloric ulcer.	Irregular in outline.	Growth lesser curvature.	
300	29	F.	? Carcinoma of stomach.	Large atonic stomach; nil else.	Operation six months later; large mass involving anterior wall.	
342	66	F.	Carcinoma of stomach.	Gastric cavity obliterated, except along greater curvature.	Post-mortem; massive carcinoma involving whole stomach.	
366	55	F.	Abdom. tumour.	Obliteration of all the cavity, except greater curvature.	Large mass extending from lesser curvature.	
380	43	M.	Duodenal ulcer.	Stomach cavity small and irregular; back pressure oesophageal dilatation.	Stomach one mass of growth.	
385	55	F.	Carcinoma of stomach.	Nil abnormal made out.	Growth lesser curvature, size of Tangerine.	
403	50	M.	—	Irregularity of pyloric portion.	Post-mortem; carcinoma.	
469	66	M.	? Carcinoma.	Cavity nearly obliterated.	Large growth; inoperable.	
592	52	F.	Carcinoma of ascending colon.	Cavity nearly obliterated; right side diaphragm pushed up.	Growth involving colon and stomach, secondary in liver.	
614	60	M.	? Carcinoma of pylorus.	Irregularities near pylorus; no obstruction.	Carcinoma of pylorus.	
776	47	M.	? Gastric ulcer; ? pyloric stenosis.	Marked inroad of pyloric portion; retained food.	Carcinoma of lesser curvature and pylorus.	Patient died 36 hours later, cause unknown.

CLASS VI. (See p. 69.) PYLORIC OBSTRUCTION.

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
10 35 F.	Dyspepsia.	Complete atony; bismuth seen in stomach for 5 days after examination.	Pyloric obstruction (cicatricial); gastro-jejuno-ostomy.	Not cured.
20 52 M.	? Carcinoma.	Normal stomach, full of fluid; bismuth in stomach after 24 hours.	Carcinoma of pylorus with ulceration.	
24 50 M.	Gastric ulcer.	Slight delay in emptying.	Ulcer of lesser curvature and thickening of pylorus; gastro-jejunostomy.	Cured. See also under Class IV.
29 54 M.	? Carcinoma of stomach.	Marked atony; retained fluid; slight delay.	Ulcer surrounding pylorus; gastro-jejunostomy.	
31 55 F.	? Carcinoma of stomach.	Retained food; tone perfect; bismuth retained 24 hours.	Extensive carcinoma involving pylorus; gastro-jejunostomy.	Patient cured.
41 32 F.	—	Hour-glass, partly spasmodic; peristalsis of upper sac; delayed emptying of lower sac.	Pyloric thickening; no ulcer of body.	Not cured; patient per- sisted in vomit- ing from upper sac.
56 44 F.	? Gastric ulcer.	Visceroptosis; delay in emptying; marked peristalsis.	Thickening of pylorus; gastro-jejunostomy.	Cured.
65 25 F.	? Gastric ulcer.	Some delay in emptying, in spite of excessive peristalsis; some visceroptosis.	Thickening about pylorus; gastro-jejunostomy.	Relieved by operation.
72 61 F.	Gastric ulcer.	Extreme atony; marked peristalsis; delayed emptying (24 hours).	Thickening about pylorus; gastro-jejunostomy.	Cured.
80 46 M.	? Pyloric obstruction.	Retained food; perfect tone; defective peris- talsis; delayed emptying (24 hours).	Large mass about pylorus.	
88 47 F.	Carcinoma of pylorus.	Stomach atonic; marked delay emptying (24 hours).	Growth at pylorus; huge stomach; gastro- jejunostomy.	'Cured' 2 years later.
94 32 M.	Dilat. stomach.	Definite delay in emptying (12 hours); some atony.	Thickening of pylorus.	Cured.
98 43 F.	? Gastric ulcer.	Large atonic stomach with retained food; slight delay in emptying; marked peristalsis.	Adhesions along lesser curvature and pylorus; ? as to obstruction.	Cured.
126 46 M.	Duodenal ulcer.	Nil, except rather active peristalsis; stomach emptying rapidly.	9 months later pyloric obstruction found.	Rapid empty- ing noted in early stage.
175 42 M.	Gastric ulcer.	Definite delay in emptying; some atony.	Ulcer of pylorus; adhesions to liver.	Cured.
179 34 F.	—	Very atonic stomach; active peristalsis; slight delay emptying.	Pyloric thickening and ulcer; stomach very large.	Cured.

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
192 51 M.	—	Retained food; delay in emptying.	Growth about pylorus and liver.	
201 48 M.	? Duod. ulcer; ? malignant.	Definite delay (24 hours); stomach normal; retained food; peristalsis not active.	Cicatrix of pylorus; gastro-jejunostomy.	Cured.
205 36 M.	Duodenal ulcer.	Stomach normal; violent peristalsis occasionally; slight delayed emptying (10 hours).	Old cicatrices about pylorus and duodenum; gastro-jejunostomy.	Patient died 3 days after operation; no cause known.
208 35 M.	Pyloric obstruction.	Retained food 24 hours; perfect tone; peristalsis absent.	Ring of ulceration round pylorus.	Cured.
217 36 F.	Carcinoma of stomach.	Huge atonic stomach; definite delay in emptying (24 hours).	Huge stomach; growth around pylorus.	
220 49 M.	Duodenal ulcer.	Stomach normal; very active peristalsis; occasionally some delay emptying.	Adhesion of pylorus and duodenum to gall-bladder; ? pyloric obstruction.	
226 29 M.	Duodenal ulcer.	Stomach toneless; definite delay (24 hours).	Thickening of pylorus and scarring of duodenum.	Cured.
230 39 F.	? Pyloric stenosis.	Hour-glass stomach and definite delay in emptying lower sac.	Ring of ulceration greater curvature and constriction of pylorus.	
239 40 F.	Gastric ulcer.	Funnel-shaped hour-glass; inverted peristalsis in lower sac; delay in emptying (24 hours).	Multiple scars forming trilobular stomach; growth at pylorus.	
250 49 M.	Dilat. stomach.	Huge distended stomach full of food; marked delay (48 hours).	Large growth of pylorus.	
269 40 F.	? Dilated stomach; pyloric stenosis.	Complete atony; delay in emptying (24 hours); incomplete hour-glass.	Cicatrices about pylorus and smaller one on greater curvature.	Cured.
272 43 M.	Duodenal ulcer.	Stomach, normal, but occasionally active peristalsis.	Adhesions and glands malignant? about pylorus.	
293 35 F.	Dilat. stomach.	Extreme atony; delay emptying.	Growth at pylorus; gastro-jejunostomy.	Patient cured (6 months).
294 43 M.	Gastric ulcer.	Slight atony; occasional violent waves of peristalsis; no marked delay emptying.	Thickening pylorus; gastro-jejunostomy.	Cured.
295 50 M.	? Carcinoma of stomach.	Definite delay in emptying; occasional violent peristalsis; inroads of growth at pylorus.	Growth about pylorus.	
309 56 M.	? hour-glass.	Retained fluid; perfect tone; peristalsis absent; definite delay (24 hours).	Large growth around pylorus; gastro-jejunostomy.	

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
313 34 M.	—	Retained fluid; stomach normal; no peristalsis; delay emptying (24 hours).	Cicatrices; stenosis of pylorus.	
320 37 M.	Gastric ulcer.	Rather active peristalsis; some atony; delay in emptying.	Mass adherent to pylorus.	Stomach the flabbiest surgeon had handled.
330 47 M.	Pyloric obstruction.	Retained food; active peristalsis, but no marked delay in emptying (10 hours).	Thickening about pylorus.	
335 39 F.	? Gastric ulcer; ? carcinoma.	Hour-glass; secretion into upper sac; delay in emptying lower sac (24 hours).	Typical hour-glass stomach; ulceration and cicatrization of pylorus.	Secretion into upper sac.
338 40 M.	Dilat. stomach.	Atonic stomach; delay in emptying (24 hours).	Thickening of pylorus.	
340 65 M.	Dilat. stomach.	Atonic stomach; retained food; delay emptying (24 hours).	Large cicatrix.	
342 42 M.	Dilat. stomach; ? carcinoma.	Retained food; occasional active peristalsis; inroad near pylorus; slight delay emptying.	Inoperable carcinoma of pylorus.	
355 65 M.	? Gastric carcinoma; ? gastric ulcer.	Delay in emptying; irregularities near pylorus.	Pyloric thickening; adhesions to liver.	
359 60 F.	Carcinoma of stomach.	Atonic stomach; retained food; delay in emptying.	Pyloric thickening; huge stomach.	Patient died 1 week later, ? cause.
370 M.	—	Retained food; violent peristalsis; no atony.	Pyloric obstruction; large stomach.	
373 49 F.	Pyloric obstruction.	Atonic; delay in emptying (24 hours).	Carcinoma of pylorus; gastro-jejunostomy.	
375 41 F.	Pyloric obstruction.	Atonic; delay in emptying (24 hours).	Pyloric obstruction; gastro-jejunostomy.	
383 59 M.	Abdominal tumour.	Atonic; delay in emptying; no inroads.	Growth of pylorus.	
399 33 M.	Gastralgia.	Atonic; retained food; slight delay in emptying (6 hours).	Large ulcer pylorus.	
410 63 M.	?	Retained food; delayed emptying; tone perfect.	Ulcer pylorus; gastro-jejunostomy.	
411 36 M.	Gastropnoia.	Delay in emptying (10 hours).	Thickening pylorus.	
421 58 M.	Duodenal ulcer.	Irregularities near pylorus; delay in emptying (24 hours); tone fair.	Carcinoma of pylorus.	
422 57 F.	Pyloric obstruction.	Irregularities; delay in emptying (24 hours).	Carcinoma of pylorus; thin stomach walls.	

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
460 57 M.	Pyloric obstruction. growth?	Delay in emptying (6 days); stomach atonic.	Growth of pylorus and duodenum.	
465 39 F.	Gastric ulcer.	Incomplete hour-glass; delayed emptying lower sac.	Pyloric obstruction; old cicatrix; anterior wall of stomach.	
470 36 F.	Gastric ulcer.	Atonic; slight delay in emptying; active peristalsis.	Pyloric ulcer; gastro-jejunosotomy.	Cured.
487 21 F.	Dilated stomach.	Delayed emptying; atonic stomach; retained food.	Growth of pylorus; gastro-jejunosotomy.	
490 24 F.	Dilated stomach.	Slight delayed emptying; spasm middle stomach.	Large stomach; cicatrix near pylorus and also ulcer on greater curvature; gastro-jejunosomy.	
492 31 F.	Neurosis.	Hour-glass, and delayed emptying lower sac (24 hours).	Hour-glass; adhesions to liver; pyloric obstruction.	
494 48 M.	Duodenal ulcer.	Stomach displaced; retained food; delayed emptying.	Large saddle-shaped ulcer about pylorus; adhesions to liver.	
500 46 M.	Gastritis.	Atonic; delayed emptying (24 hours).	Growth of pylorus.	
512 47 F.	Pyloric obstruction.	Atonic; retained food; delayed emptying (24 hours).	Thickening about pylorus; gastro-jejunosotomy.	
523 42 F.	Gastric ulcer; dilated stomach.	Atonic stomach; retained food; delayed emptying.	Thickening about pylorus; gastro-jejunosotomy.	
531 42 M.	—	Atonic; retained food; delayed emptying (24 hours).	Stomach very large; cicatrix about pylorus.	
573 18 F.	? Gastric ulcer.	Hour-glass (cicatricial); hypersecretion to upper sac; delayed emptying of lower sac.	'As though string had been tied round stomach'; also pyloric ulcer	Hypersecretion with pyloric ulcer.
580 43 F.	Pyloric obstruction; dilated stomach.	Atonic; retained food; delayed emptying.	Thickening of pylorus; malignant.	
582 29 M.	?	Delayed emptying (6 hours); stomach atonic.	Thickening of pylorus.	
583 54 M.	Abdominal tumour.	Atonic; delayed emptying (24 hours); no irregularities.	Carcinoma of pylorus.	
584 42 F.	Pyloric obstruction.	Atonic; delayed emptying (24 hours).	Post-mortem; thickened pylorus.	
585 43 M.	Duodenal ulcer.	Retained food; some delay in emptying (8 hours).	Thickened pylorus; gastro-jejunosotomy.	

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
585a44 F.	Pyloric obstruction.	Stomach normal; active peristalsis; hypersecretion; slight delay in emptying?	Adhesions and cicatrix about pylorus and duodenum.	
586a62 F.	—	Atonic; delay in emptying (24 hours).	Carcinoma of pylorus.	
590 21 F.	Dilated stomach.	Atonic stomach; active peristalsis; excessive secretion.	Growth at pylorus; gastro-jejunosomy.	
592 33 M.	Gastralgia.	Atonic stomach; delay in emptying; retained food.	Large ulcer at pylorus; stomach large; gastro-jejunosomy.	
600 40 F.	Visceroptosis.	Atonic; delay in emptying.	Thickening of pylorus; carcinoma?	
687 60 M.	? Carcinoma of stomach.	Atonic; delay in emptying (24 hours); retained food.	Cicatrix of pylorus.	
682 27 M.	Pyloric tumour.	Tone good; delay in emptying; no peristalsis; obliteration of pylorus.	Carcinoma of pylorus.	
752 40 M.	? Duodenal ulcer.	Atonic; retained food (24 hours).	Ulcer of pylorus; adhesions.	
753 25 M.	Gastric ulcer.	Atonic; retained food; displaced pyloric portion; active peristalsis.	Large ulcer pylorus.	
545 23 M.	Hodgkin's disease; pyloric obstruction.	Stomach normal; occasional excessive peristalsis; excessive secretion; gave off little CO ₂ .	Carcinoma at pylorus; many secondary glands.	
664 28 M.	Gastric ulcer; pyloric obstruction.	Stomach normal; occasional excessive peristalsis; excessive secretion; gave off little CO ₂ ; retained food.	Large growth of pyloric region and secondary glands.	
689 30 F.	Gastric ulcer.	Stomach atonic; retained food; delayed emptying (24 hours).	Pyloric obstruction marked; old ulcer.	Patient was examined 2 months before and very slight, if any, delayed emptying was noted.
698 45 M.	? Carcinoma of pylorus.	Retained food; tone perfect; peristalsis absent.	Carcinoma of pylorus.	
713 32 M.	Gastric ulcer.	Delayed emptying; spasm middle.	Ulcers about pylorus and cicatrices.	
753b48 F.	Carcinoma of stomach?	Retained food; stomach atonic; delayed emptying (24 hours).	Carcinoma of pylorus fungating into stomach.	
756 35 F.	Old gastro-jejunosomy.	Stoma not working; some delay emptying; looks like pyloric obstruction.	No gastro-jejunosomy (had ever been performed; cicatrix of pylorus; gastro-jejunosomy).	
764 47 F.	Pyloric obstruction.	Retained food; slight delay emptying (10 hours).	Pyloric obstruction; gastro-jejunosomy.	

CLASS VII. (See p. 89.) ADHESIONS.

No.	Age.	Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
151	38	F.	Neurasthenia.	Stomach atonic; held down to colon by adhesions; transverse colon fixed in pelvis.	Old appendix; adhesions of colon.	Not cured. 18 months later much better.
251	10	M.	T.B. peritonitis.	Bilocular stomach; food retained in many pockets of small intestines.	Tuberculosis; peritonitis; multiple adhesions.	Relieved.
408	61	F.	Gastric ulcer.	Bands divided stomach, and as in Case 251.	Multiple adhesions; old ulcer below cardiac orifice.	
472	60	M.	Old perforated duodenal ulcer.	Adhesions to liver, etc.	Adhesions anterior wall and liver.	
479	42	F.	Old strangulated hernia; ? adhesions.	Stomach bound down in pelvis.	Adhesions small intestine, stomach and colon.	
591	28	M.	Dyspepsia.	Nil, except stomach displaced to right.	Appendix adhesions on right side; stomach normal.	Long appendix fixed up towards right hypochondrial region.
699	56	M.	? Carcinoma of stomach or oesophagus.	Stomach cavity represented by 3 sacs, very small, bound to liver; back pressure oesophageal dilatation.	Mass of adhesions stomach to liver; anterior gastro-jejunostomy.	

CLASS VIII. (See p. 92).*

POST OPERATIVE.

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
10 35 F.	Old Gastro-jejunostomy (see Class VI.).	No increase in tone; food still delayed in stomach longer than 24 hours.	—	
19 55 F.	Pyloroplasty.	Hour-glass condition; no obstruction, stoma working perfectly.	—	Patient unrelieved.
26 19 F.	Perforated gastric ulcer.	Food through stoma quite freely.	—	
35 32 F.	Old gastro-jejunostomy (see Class IV.).	Stoma working perfectly.	—	
41 32 F.	(See Class VI.).	Hour-glass marked, but stoma works perfectly when food reaches it.	Nil found to account for hour-glass.	Patient persisted in vomiting from upper sac.
44 25 F.	Gastro-jejunostomy.	Obstruction in duodenum.	Adhesions.	Patient spoken to severely and completely cured.
206 23 F.	Old gastro-jejunostomy.	Stoma working perfectly.	—	Unrelieved.
207 30 F.	Old gastro-jejunostomy. 1908.	Gastro-jejunostomy working perfectly; marked spasm middle of stomach forming hour-glass.	Marked cicatrix above stoma; gastro-gastrotomy.	
209 31 F.	Old gastro-jejunostomy for hour-glass.	Stoma from upper sac working perfectly, but food retained in lower sac 24 hours after.	Well-marked pyloric obstruction; lower sac excised.	Patient cured.
257 49 F.	Old gastro-jejunostomy.	Adhesions forming hour-glass; stoma working perfectly.	Adhesions broken down.	Cured.
263 41 F.	5 weeks after gastro-jejunostomy.	Stoma working perfectly.	—	
266 43 M.	Old gastro-jejunostomy.	Hour-glass stomach; well-marked; stoma working perfectly.	Hour-glass, with active ulcer.	Cured by gastrotomy; a small ulcer of body had been noted at time of first operation.
357 52 F.	Old gastro-jejunostomy to upper sac of hour-glass.	Delayed emptying of lower sac; stoma from upper sac works well.	Thickening of pylorus; lower sac excised.	Cured.

No.	Age.	Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
363	41	F.	Old gastro-jejunostomy.	Spasmodic hour-glass; stoma working perfectly.	Nil found to account for hour-glass.	Unrelieved.
398	38	F.	—	Hour-glass; marked vomiting from upper sec.	Nothing to account for the hour-glass; appendix removed.	Cured five weeks after operation.
416	47	M.	Old gastro-jejunostomy.	Adhesions about jejunum and stomach.	Many adhesions.	The pain appeared to be due to the contractions of the stomach on the food in the pyloric portion; relieved by operation.
419	22	F.	Old gastro-jejunostomy 2 months after operation.	Stomach working perfectly, but placed far from pylorus.	Growth of pylorus; adherent to gall-bladder; growth excised.	Cured.
470	36	F.	3 weeks after gastro-jejunostomy.	Jejunum kinked behind stomach.	Jejunum stitched up.	
471	30	F.	Gastro-jejunostomy.	Stoma not patent.	Adhesions.	
483	30	M.	Old gastro-jejunostomy.	Kink 6 inches from stoma.	Adhesions with kinking of jejunum.	
524	46	F.	Gastro-jejunostomy 5 years ago.	Stomach atonic; excessive peristalsis; stoma not working; obstruction at duodeno-jejunal flexure.	Cicatrization about stoma jejunum, and adhesions.	
539	35	M.	Old gastro-jejunostomy 1 year after operation.	Food passing freely both ways; excessive peristalsis; stoma 5 inches from pylorus.	—	
563	36	F.	Old gastro-jejunostomy 2 years ago.	Stoma working perfectly.	—	
530	47	M.	Old gastro-jejunostomy 10 months.	Adhesions blocking jejunum.	Many adhesions dissected out.	Patient died.
594	45	M.	Old gastro-jejunostomy; duodenal obstruction.	Stoma working perfectly; hypersecretion.	Adhesions anterior wall.	Cured.
609	50	F.	Gastro-jejunostomy 2 years.	Both stoma and pylorus patent.	—	
689	30	F.	Gastro-jejunostomy 6 weeks after operation.	Stoma not working; looks like pyloric obstruction.	No gastro-jejunostomy had been performed; cicatrix of pylorus.	Cured.
756	35	F.	Old gastro-jejunostomy.	Stoma not working; slight delay emptying.	No gastro-jejunostomy had been performed; cicatrix of pylorus; gastro-jejunostomy.	
757	34	M.	Old gastro-jejunostomy.	Stoma working well; slight obstruction in jejunum.	—	

CLASS V. (See p. 85.)

CARCINOMA OF THE STOMACH.

No.	Age.	Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
7	43	M.	Carcinoma of stomach?	Very excessive peristalsis; pars pylorica obliterated. No delay in emptying.	Annular growth of pylorus.	Excised growth; patient died 18 months later.
8	35	M.	Carcinoma of stomach.	Stomach invaded; small irregular cavity only left.	Advanced carcinoma; inoperable.	
9	43	M.	Carcinoma of stomach.	Inroads of growth well defined.	Inoperable carcinoma.	
21	45	M.	—	Irregularity in outline of greater curvature.	Inoperable carcinoma.	
40	37	M.	? Carcinoma.	Irregularities of outline.	Advanced carcinoma.	
42	53	F.	? Carcinoma of stomach.	Inroads of growth near pylorus; retention of food.	Carcinoma of pylorus; gastro-jejunostomy.	
48	52	F.	Carcinoma of stomach.	Esophageal obstruction at cardiac orifice; no evidence of growth of stomach.	Post-mortem six weeks later; growth of lesser curvature 5" x 6"; cardiac orifice not involved.	No contraction of cardiac orifice post-mortem.
58	54	M.	? Carcinoma of stomach; ? aneurism.	Light areas in midst of bismuth shadow.	Inoperable carcinoma; posterior wall chiefly.	Patient died 3 months later.
64	39	M.	? Carcinoma of stomach.	Irregular in outline.	Advanced carcinoma.	
170	58	M.	? Carcinoma of stomach.	Inroads of growth giving hour-glass appearance.	Inoperable carcinoma.	
233	53	F.	? Carcinoma of stomach.	Definite irregularities of outline.	Mass of growth in lesser curvature.	
289	46	M.	? Pyloric obstruction.	Stomach normal; shadows well seen in duodenum; active peristalsis.	Plaque of growth on lesser curvature; duodenum not explored.	No evidence of growth of stomach.

No.	Age.	Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
297	34	M.	Pyloric ulcer.	Irregular in outline.	Growth lesser curvature.	
300	29	F.	? Carcinoma of stomach.	Large atonic stomach; nil else.	Operation six months later; large mass involving anterior wall.	
342	66	F.	Carcinoma of stomach.	Gastric cavity obliterated, except along greater curvature.	Post-mortem; massive carcinoma involving whole stomach.	
366	55	F.	Abdom. tumour.	Obliteration of all the cavity, except greater curvature.	Large mass extending from lesser curvature.	
380	43	M.	Duodenal ulcer.	Stomach cavity small and irregular; back pressure oesophageal dilatation.	Stomach one mass of growth.	
385	55	F.	Carcinoma of stomach.	Nil abnormal made out.	Growth lesser curvature, size of Tangerine.	
403	50	M.	—	Irregularity of pyloric portion.	Post-mortem; carcinoma.	
469	66	M.	? Carcinoma.	Cavity nearly obliterated.	Large growth; inoperable.	
582	52	F.	Carcinoma of ascending colon.	Cavity nearly obliterated; right side diaphragm pushed up.	Growth involving colon and stomach, secondary in liver.	
614	60	M.	? Carcinoma of pylorus.	Irregularities near pylorus; no obstruction.	Carcinoma of pylorus.	
776	47	M.	? Gastric ulcer; ? pyloric stenosis.	Marked inroad of pyloric portion; retained food.	Carcinoma of lesser curvature and pylorus.	Patient died 36 hours later, cause unknown.

CLASS VI. (See p. 69.) PYLORIC OBSTRUCTION.

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
10 35 F.	Dyspepsia.	Complete atony; bismuth seen in stomach for 5 days after examination.	Pyloric obstruction (cicatrical); gastro-jejunoscopy.	Not cured.
20 52 M.	? Carcinoma.	Normal stomach, full of fluid; bismuth in stomach after 24 hours.	Carcinoma of pylorus with ulceration.	
24 50 M.	Gastric ulcer.	Slight delay in emptying.	Ulcer of lesser curvature and thickening of pylorus; gastro-jejunoscopy.	Cured. See also under Class IV.
29 54 M.	? Carcinoma of stomach.	Marked atony; retained fluid; slight delay.	Ulcer surrounding pylorus; gastro-jejunoscopy.	Patient cured.
31 55 F.	? Carcinoma of stomach.	Retained food; tone perfect; bismuth retained 24 hours.	Extensive carcinoma involving pylorus; gastro-jejunoscopy.	
41 32 F.	—	Hour-glass, partly spasmodic; peristalsis of upper sac; delayed emptying of lower sac.	Pyloric thickening; no ulcer of body.	Not cured; patient per- sisted in vomit- ing from upper sac. Cured.
56 44 F.	? Gastric ulcer.	Visceroptosis; delay in emptying; marked peristalsis.	Thickening of pylorus; gastro-jejunoscopy.	Relieved by operation. Cured.
65 25 F.	? Gastric ulcer.	Some delay in emptying, in spite of excessive peristalsis; some visceroptosis.	Thickening about pylorus; gastro-jejunoscopy.	
72 61 F.	Gastric ulcer.	Extreme atony; marked peristalsis; delayed emptying (24 hours).	Thickening about pylorus; gastro-jejunoscopy.	
80 46 M.	? Pyloric obstruction.	Retained food; perfect tone; defective peristalsis; delayed emptying (24 hours).	Large mass about pylorus.	
88 47 F.	Carcinoma of pylorus.	Stomach atonic; marked delay emptying (24 hours).	Growth at pylorus; huge stomach; gastro-jejunoscopy.	'Cured' 2 years later.
94 32 M.	Dilat. stomach.	Definite delay in emptying (12 hours); some atony.	Thickening of pylorus.	Cured.
98 43 F.	? Gastric ulcer.	Large atonic stomach with retained food; slight delay in emptying; marked peristalsis.	Adhesions along lesser curvature and pylorus; ? as to obstruction.	Cured.
126 46 M.	Duodenal ulcer.	Nil, except rather active peristalsis; stomach emptying rapidly.	9 months later pyloric obstruction found.	Rapid empty- ing noted in early stage. Cured.
175 42 M.	Gastric ulcer.	Definite delay in emptying; some atony.	Ulcer of pylorus; adhesions to liver.	Cured.
179 34 F.	—	Very atonic stomach; active peristalsis; slight delay emptying.	Pyloric thickening and ulcer; stomach very large.	Cured.

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
192 51 M.	—	Retained food; delay in emptying.	Growth about pylorus and liver.	
201 48 M.	? Duod. ulcer; ? malignant.	Definite delay (24 hours); stomach normal; retained food; peristalsis not active.	Cicatrix of pylorus; gastro-jejunostomy.	Cured.
205 36 M.	Duodenal ulcer.	Stomach normal; violent peristalsis occasionally; slight delayed emptying (10 hours).	Old cicatrices about pylorus and duodenum; gastro-jejunostomy.	Patient died 3 days after operation; no cause known.
208 35 M.	Pyloric obstruction.	Retained food 24 hours; perfect tone; peristalsis absent.	Ring of ulceration round pylorus.	Cured.
217 36 F.	Carcinoma of stomach.	Huge atonic stomach; definite delay in emptying (24 hours).	Huge stomach; growth around pylorus.	
220 49 M.	Duodenal ulcer.	Stomach normal; very active peristalsis; occasionally some delay emptying.	Adhesion of pylorus and duodenum to gall-bladder; ? pyloric obstruction.	
226 29 M.	Duodenal ulcer.	Stomach toneless; definite delay (24 hours).	Thickening of pylorus and scarring of duodenum.	Cured.
230 39 F.	? Pyloric stenosis.	Hour-glass stomach and definite delay in emptying lower sac.	Ring of ulceration greater curvature and constriction of pylorus.	
239 40 F.	Gastric ulcer.	Funnel-shaped hour-glass; inverted peristalsis in lower sac; delay in emptying (24 hours).	Multiple scars forming trilobular stomach; growth at pylorus.	
250 49 M.	Dilat. stomach.	Huge distended stomach full of food; marked delay (48 hours).	Large growth of pylorus.	
269 40 F.	? Dilated stomach; pyloric stenosis.	Complete atony; delay in emptying (24 hours); incomplete hour-glass.	Cicatrices about pylorus and smaller one on greater curvature.	Cured.
272 43 M.	Duodenal ulcer.	Stomach, normal, but occasionally active peristalsis.	Adhesions and glands malignant? about pylorus.	
293 35 F.	Dilat. stomach.	Extreme atony; delay emptying.	Growth at pylorus; gastro-jejunostomy.	Patient cured (6 months).
294 43 M.	Gastric ulcer.	Slight atony; occasional violent waves of peristalsis; no marked delay emptying.	Thickening pylorus; gastro-jejunostomy.	Cured.
295 50 M.	? Carcinoma of stomach.	Definite delay in emptying; occasional violent peristalsis; inroads of growth at pylorus.	Growth about pylorus.	
309 56 M.	? hour-glass.	Retained fluid; perfect tone; peristalsis absent; definite delay (24 hours).	Large growth around pylorus; gastro-jejunostomy.	

No.	Age.	Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
313	34	M.	—	Retained fluid; stomach normal; no peristalsis; delay emptying (24 hours).	Cicatrices; stenosis of pylorus.	
320	37	M.	Gastric ulcer.	Rather active peristalsis; some atony; delay in emptying.	Mass adherent to pylorus.	Stomach the flabbiest surgeon had handled.
330	47	M.	Pyloric obstruction.	Retained food; active peristalsis, but no marked delay in emptying (10 hours).	Thickening about pylorus.	
335	39	F.	? Gastric ulcer; ? carcinoma.	Hour-glass; secretion into upper sac; delay in emptying lower sac (24 hours).	Typical hour-glass stomach; ulceration and cicatrization of pylorus.	Secretion into upper sac.
338	40	M.	Dilat. stomach.	Atonic stomach; delay in emptying (24 hours).	Thickening of pylorus.	
340	65	M.	Dilat. stomach.	Atonic stomach; retained food; delay emptying (24 hours).	Large cicatrix.	
342	42	M.	Dilat. stomach; ? carcinoma.	Retained food; occasional active peristalsis; inroad near pylorus; slight delay emptying.	Inoperable carcinoma of pylorus.	
355	65	M.	? Gastric carcinoma; ? gastric ulcer.	Delay in emptying; irregularities near pylorus.	Pyloric thickening; adhesions to liver.	
359	60	F.	Carcinoma of stomach.	Atonic stomach; retained food; delay in emptying.	Pyloric thickening; huge stomach.	Patient died 1 week later, ? cause.
370	M.	—	—	Retained food; violent peristalsis; no atony.	Pyloric obstruction; large stomach.	
373	49	F.	Pyloric obstruction.	Atonic; delay in emptying (24 hours).	Carcinoma of pylorus; gastro-jejunostomy.	
375	41	F.	Pyloric obstruction.	Atonic; delay in emptying (24 hours).	Pyloric obstruction; gastro-jejunostomy.	
383	59	M.	Abdominal tumour.	Atonic; delay in emptying; no inroads.	Growth of pylorus.	
399	33	M.	Gastralgia.	Atonic; retained food; slight delay in emptying (6 hours).	Large ulcer pylorus.	
410	63	M.	?	Retained food; delayed emptying; tone perfect.	Ulcer pylorus; gastro-jejunostomy.	
411	36	M.	Gastropnoia.	Delay in emptying (10 hours).	Thickening pylorus.	
421	58	M.	Duodenal ulcer.	Irregularities near pylorus; delay in emptying (24 hours); tone fair.	Carcinoma of pylorus.	
422	57	F.	Pyloric obstruction.	Irregularities; delay in emptying (24 hours).	Carcinoma of pylorus; thin stomach walls.	

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
460 57 M.	Pyloric obstruction. growth?	Delay in emptying (6 days); stomach atonic.	Growth of pylorus and duodenum.	
465 39 F.	Gastric ulcer.	Incomplete hour-glass; delayed emptying lower sac.	Pyloric obstruction; old cicatrix; anterior wall of stomach.	
470 36 F.	Gastric ulcer.	Atonic; slight delay in emptying; active peristalsis.	Pyloric ulcer; gastro-jejunosotomy.	Cured.
487 21 F.	Dilated stomach.	Delayed emptying; atonic stomach; retained food.	Growth of pylorus; gastro-jejunosotomy.	
490 24 F.	Dilated stomach.	Slight delayed emptying; spasm middle stomach.	Large stomach; cicatrix near pylorus and also ulcer on greater curvature; gastro-jejunosomy.	
492 31 F.	Neurosis.	Hour-glass, and delayed emptying lower sac (24 hours).	Hour-glass; adhesions to liver; pyloric obstruction.	
494 48 M.	Duodenal ulcer.	Stomach displaced; retained food; delayed emptying.	Large saddle-shaped ulcer about pylorus; adhesions to liver.	
500 46 M.	Gastritis.	Atonic; delayed emptying (24 hours).	Growth of pylorus.	
512 47 F.	Pyloric obstruction.	Atonic; retained food; delayed emptying (24 hours).	Thickening about pylorus; gastro-jejunosomy.	
523 42 F.	Gastric ulcer; dilated stomach.	Atonic stomach; retained food; delayed emptying.	Thickening about pylorus; gastro-jejunosomy.	
531 42 M.	—	Atonic; retained food; delayed emptying (24 hours).	Stomach very large; cicatrix about pylorus.	
573 18 F.	? Gastric ulcer.	Hour-glass (cicatrical); hypersecretion to upper sac; delayed emptying of lower sac.	'As though string had been tied round stomach'; also pyloric ulcer.	Hypersecretion with pyloric ulcer.
580 43 F.	Pyloric obstruction; dilated stomach.	Atonic; retained food; delayed emptying.	Thickening of pylorus; malignant.	
582 29 M.	?	Delayed emptying (6 hours); stomach atonic.	Thickening of pylorus.	
583 54 M.	Abdominal tumour.	Atonic; delayed emptying (24 hours); no irregularities.	Carcinoma of pylorus.	
584 42 F.	Pyloric obstruction.	Atonic; delayed emptying (24 hours).	Post-mortem; thickened pylorus.	
585 43 M.	Duodenal ulcer.	Retained food; some delay in emptying (8 hours).	Thickened pylorus; gastro-jejunosotomy.	

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
585a44 F.	Pyloric obstruction.	Stomach normal; active peristalsis; hypersecretion; slight delay in emptying?	Adhesions and cicatrix about pylorus and duodenum.	
586a62 F.	—	Atonic; delay in emptying (24 hours).	Carcinoma of pylorus.	
590 21 F.	Dilated stomach.	Atonic stomach; active peristalsis; excessive secretion.	Growth at pylorus; gastro-jejunosotomy.	
592 33 M.	Gastralgia.	Atonic stomach; delay in emptying; retained food.	Large ulcer at pylorus; stomach large; gastro-jejunosotomy.	
600 40 F.	Visceroptosis.	Atonic; delay in emptying.	Thickening of pylorus; carcinoma?	
687 60 M.	? Carcinoma of stomach.	Atonic; delay in emptying (24 hours); retained food.	Cicatrix of pylorus.	
682 27 M.	Pyloric tumour.	Tone good; delay in emptying; no peristalsis; obliteration of pylorus.	Carcinoma of pylorus.	
752 40 M.	? Duodenal ulcer.	Atonic; retained food (24 hours).	Ulcer of pylorus; adhesions.	
753 25 M.	Gastric ulcer.	Atonic; retained food; displaced pyloric portion; active peristalsis.	Large ulcer pylorus.	
545 23 M.	Hodgkin's disease; pyloric obstruction.	Stomach normal; occasional excessive peristalsis; excessive secretion; gave off little CO ₂ .	Carcinoma at pylorus; many secondary glands.	
664 28 M.	Gastric ulcer; pyloric obstruction.	Stomach normal; occasional excessive peristalsis; excessive secretion; gave off little CO ₂ ; retained food.	Large growth of pyloric region and secondary glands.	
689 30 F.	Gastric ulcer.	Stomach atonic; retained food; delayed emptying (24 hours).	Pyloric obstruction marked; old ulcer.	Patient was examined 2 months before and very slight, if any, delayed emptying was noted.
698 45 M.	? Carcinoma of pylorus.	Retained food; tone perfect; peristalsis absent.	Carcinoma of pylorus.	
713 32 M.	Gastric ulcer.	Delayed emptying; spasm middle.	Ulcers about pylorus and cicatrices.	
753b48 F.	Carcinoma of stomach?	Retained food; stomach atonic; delayed emptying (24 hours).	Carcinoma of pylorus fungating into stomach.	
756 35 F.	Old gastro-jejunosotomy.	Stoma not working; some delay emptying; looks like pyloric obstruction.	No gastro-jejunosotomy had ever been performed; cicatrix of pylorus; gastro-jejunosotomy.	
764 47 F.	Pyloric obstruction.	Retained food; slight delay emptying (10 hours).	Pyloric obstruction; gastro-jejunosotomy.	

CLASS VII. (See p. 89.)
ADHESIONS.

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
151 38 F.	Neurasthenia.	Stomach atonic; held down to colon by adhesions; transverse colon fixed in pelvis.	Old appendix; adhesions of colon.	Not cured. 18 months later much better.
251 10 M.	T.B. peritonitis.	Bilocular stomach; food retained in many pockets of small intestines.	Tuberculosis; peritonitis; multiple adhesions.	Relieved.
408 61 F.	Gastric ulcer.	Bands divided stomach, and as in Case 251.	Multiple adhesions; old ulcer below cardiac orifice.	
472 60 M.	Old perforated duodenal ulcer.	Adhesions to liver, etc.	Adhesions anterior wall and liver.	
479 42 F.	Old strangulated hernia; ? adhesions.	Stomach bound down in pelvis.	Adhesions small intestine, stomach and colon.	
591 28 M.	Dyspepsia.	Nil, except stomach displaced to right.	Appendix adhesions on right side; stomach normal.	Long appendix fixed up towards right hypochondrial region.
699 56 M.	? Carcinoma of stomach or oesophagus.	Stomach cavity represented by 3 sacs, very small, bound to liver; back pressure oesophageal dilatation.	Mass of adhesions stomach to liver; anterior gastro-jejunostomy.	

CLASS VIII. (See p. 92.)

POST OPERATIVE.

No. Age. Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
10 35 F.	Old Gastro-jejunostomy (see Class VI.).	No increase in tone; food still delayed in stomach longer than 24 hours.	—	
19 55 F.	Pyloroplasty.	Hour-glass condition; no obstruction, stoma working perfectly.	—	Patient unrelieved.
26 19 F.	Perforated gastric ulcer.	Food through stoma quite freely.	—	
35 32 F.	Old gastro-jejunostomy (see Class IV.).	Stoma working perfectly.	—	
41 32 F.	(See Class VI.).	Hour-glass marked, but stoma works perfectly when food reaches it.	Nil found to account for hour-glass.	Patient per- sisted in vomit- ing from upper sac.
44 25 F.	Gastro-jejunostomy.	Obstruction in duodenum.	Adhesions.	Patient spoken to severely and completely cured.
206 23 F.	Old gastro-jejunostomy.	Stoma working perfectly.	—	Unrelieved.
207 30 F.	Old gastro-jejunostomy. 1908.	Gastro-jejunostomy working perfectly; marked spasm middle of stomach forming hour-glass.	Marked cicatrix above stoma; gastro-gastros- tomy.	Patient cured.
209 31 F.	Old gastro-jejunostomy for hour-glass.	Stoma from upper sac working perfectly, but food retained in lower sac 24 hours after.	Well-marked pyloric obstruction; lower sac excised.	Patient cured.
257 49 F.	Old gastro-jejunostomy.	Adhesions forming hour-glass; stoma working perfectly.	Adhesions broken down.	Cured.
263 41 F.	5 weeks after gastro- jejunostomy.	Stoma working perfectly.	—	Cured by gas- troplasty; a small ulcer of body had been noted at time of first opera- tion.
266 43 M.	Old gastro-jejunostomy.	Hour-glass stomach; well-marked; stoma work- ing perfectly.	Hour-glass, with active ulcer.	Cured.
357 52 F.	Old gastro-jejunostomy to upper sac of hour-glass.	Delayed emptying of lower sac; stoma from upper sac works well.	Thickening of pylorus; lower sac excised.	

No.	Age.	Sex.	Clinical Diagnosis.	X-ray Findings.	Operative Findings.	Remarks.
363	41	F.	Old gastro-jejunoostomy.	Spasmodic hour-glass; stoma working perfectly.	Nil found to account for hour-glass.	Unrelieved.
398	38	F.	—	Hour-glass; marked vomiting from upper sac.	Nothing to account for the hour-glass; appendix removed.	Cured five weeks after operation.
416	47	M.	Old gastro-jejunoostomy.	Adhesions about jejunum and stomach.	Many adhesions.	The pain appeared to be due to the contractions of the stomach on the food in the pyloric portion; relieved by operation.
419	22	F.	Old gastro-jejunoostomy 2 months after operation.	Stomach working perfectly, but placed far from pylorus.	Growth of pylorus; adherent to gall-bladder; growth excised.	Cured.
470	36	F.	3 weeks after gastro-jejunoostomy.	Jejunum kinked behind stomach.	Jejunum stitched up.	
471	30	F.	Gastro-jejunoostomy.	Stoma not patent.	Adhesions.	
483	30	M.	Old gastro-jejunoostomy.	Kink 6 inches from stoma.	Adhesions with kinking of jejunum.	
524	46	F.	Gastro-jejunoostomy 5 years ago.	Stomach atonic; excessive peristalsis; stoma not working; obstruction at duodeno-jejunal flexure.	Cicatrization about stoma jejunum, and adhesions.	
539	35	M.	Old gastro-jejunoostomy 1 year after operation.	Food passing freely both ways; excessive peristalsis; stoma 5 inches from pylorus.	—	
563	36	F.	Old gastro-jejunoostomy 2 years ago.	Stoma working perfectly.	—	
530	47	M.	Old gastro-jejunoostomy 10 months.	Adhesions blocking jejunum.	Many adhesions dissected out.	Patient died.
594	45	M.	Old gastro-jejunoostomy; duodenal obstruction.	Stoma working perfectly; hypersecretion.	Adhesions anterior wall.	Cured.
609	50	F.	Gastro-jejunoostomy 2 years.	Both stoma and pylorus patent.	—	
689	30	F.	Gastro-jejunoostomy 6 weeks after operation.	Stoma not working; looks like pyloric obstruction.	No gastro-jejunoostomy had been performed; cicatrix of pylorus.	Cured.
756	35	F.	Old gastro-jejunoostomy.	Stoma not working; slight delay emptying.	No gastro-jejunoostomy had been performed; cicatrix of pylorus; gastro-jejunoostomy.	
757	34	M.	Old gastro-jejunoostomy.	Stoma working well; slight obstruction in jejunum.	—	

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